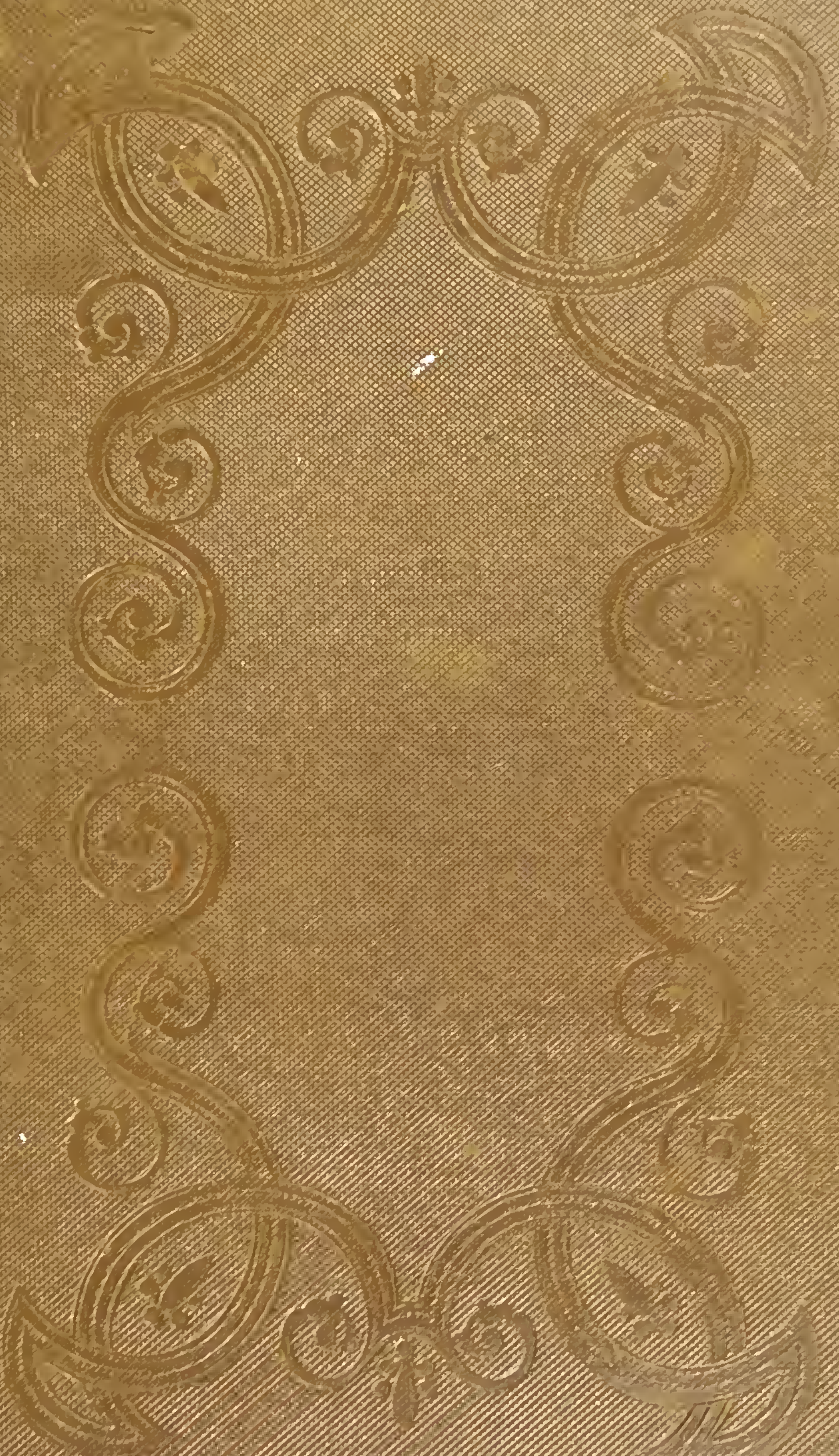

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J. G. Swayne

THE

PATHOLOGY AND DIAGNOSIS

OF

DISEASES OF THE CHEST,

COMPRISING

A Rational Exposition of their Physical Signs,

WITH AN APPENDIX;
CONTAINING VARIOUS OPINIONS AND EXPERIMENTS ON THE MOTIONS
AND SOUNDS OF THE HEART, AND ON THE BRONCHI.

BY

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HOSPITAL, LONDON UNIVERSITY COLLEGE, &c.

Fourth Edition,

MUCH ENLARGED AND ILLUSTRATED BY PLATES AND TABLES.

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TO

NEILL ARNOTT, M.D., F.R.S.,

PHYSICIAN EXTRAORDINARY TO THE QUEEN, &c.

My Dear Sir,

You have rendered inestimable service to the science and art of Medicine, by simplifying the physical laws on which they are in a great measure founded. You have earned the gratitude of men by the free gift of discoveries which may vastly contribute to their bodily comfort and health: and your active benevolence now engages you in devising plans for their moral and intellectual improvement. In dedicating this work to you, I would record at once my high sense of your good offices for our profession and for mankind at large, and my thankful acknowledgement of many acts of friendship done to myself.

I remain, with much esteem,
Most sincerely yours,
C. J. B. WILLIAMS.

October 15, 1840.

PREFACE.

More than a year has elapsed since the last edition of this work was out of print. It had been my intention to condense it into a Manual of Diagnosis of Pectoral Diseases, and to arrange in a larger work on the Nature, Causes, Distinction, and Treatment, materials which have been accumulating during many years of experience and study in Diseases of the Chest. But the important and responsible duties of teacher of practical and clinical medicine having engrossed nearly all my time, not engaged in practice, I have been obliged to postpone this task, and to complete a fourth edition on the former plan. In its present enlarged form it may have somewhat outgrown its original object; and I fear it will equally disappoint those who want only to know "how to use the stethoscope," and those who look into books only to find a remedy. But the improving standard of medical education, as well as the favourable reception of former editions, gives me reason to hope that many students and young practitioners will not be content with a smattering on the subjects of this work; but will hail in its increased size an extension of the limits of sound knowledge, which, when properly mastered and applied, is useful power.

The extensive additions made in the present edition have rendered necessary some alterations in the arrangement; that chiefly adopted being the same as that of my lectures as published in the Medical Gazette, from which also much of the new matter is taken. I trust that the engravings will be found useful improvements, and that some readers may derive advantage from the new tabular views of signs and symptoms. I have ventured to propose more simple and concise terms to designate the phenomena of auscultation; but aware of the inconvenience of any change in nomenclature, I have generally retained that hitherto employed.

The favourable reception of former editions of this work in this and in foreign countries, confirms me in the conviction, that the only fundamental mode of studying *auscultation* is through a knowledge of those facts and laws in pathology and in physics of which the phenomena of auscultation are examples. I have not the same confidence in adverting to the rational views which I have given of the *general symptoms;* but I think that the time is come when we should at least endeavour to make our improved physiological and pathological knowledge more available in practice, and connect with it, in an intelligible order, those multitudinous facts which observation has revealed.

7, *Holles-street, Cavendish-square.*
Oct. 15*th*, 1840.

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Pl.

Fig. 1.

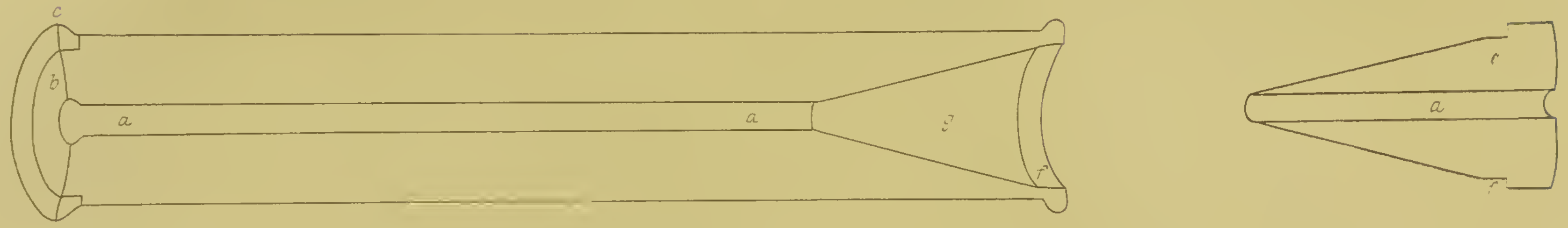
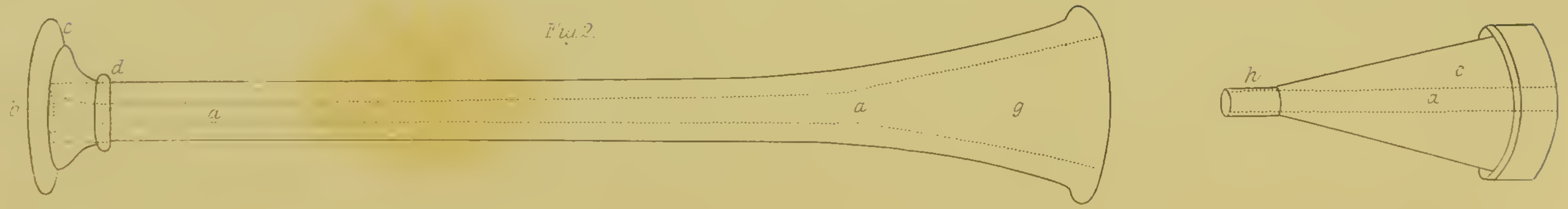


Fig. 2.



EXPLANATION OF PLATE I.

Figures 1 and 2 exhibit two of the best forms of stethoscope.

Fig. 1. is a section of a cylinder of any light rigid wood, having in it a central canal, *a*, terminating at one end by the conical excavation *g,* into which fits the plug by the flute joint *e*, *f*. The other end is slightly hollowed at *b*, and enlarged by a rim, *c*, to fit comfortably to the ear.

Fig. 2 is the trumpet-shaped stethoscope, which is generally preferable on account of its lightness. The ear-piece, *c*, *b*, slips on and off for the sake of portability. The conical stopper, *e*, fits into *g*, and holds by the short tube of ivory or boxwood, *h*.

The best material is stained deal or cedar—length about eight inches; diameter of the base of the cone, an inch and a half—of the central canal, three-eighths of an inch. The ear end should be made to fit comfortably to the ear; and from the varying shape of this feature it is impossible to describe one suitable to all cases. The edges of the other end should be sufficiently rounded not to hurt the chest of the patient.

After trying a great many modifications of this instrument, I am convinced that those above described are preferable for common purposes. I have contrived some which transmit the sounds with greater intensity, but their construction is not very simple, and their superiority not equivalent to this disadvantage. The only improvement that I shall mention as really useful for some purposes, is to cover the excavated end with a thin sheet of India-rubber drawn and cemented tight across the hollow, like a drum. With the instrument so prepared I find that I can hear the pectoral sounds through articles of clothing, nearly as well as if the chest were exposed. It is thus useful in morning consultations, and where undressing may be inconvenient. The membrane operates, I presume, by confining the column of air, and preventing the aberrations of sound between the clothes and the edge of the instrument, as adverted to at page 41. On trying, on the principle of Drs. Budd and Cowan, a thin plate of wood in the same situation, I found the transmission of the sounds very much impaired. The pectoral sounds may be increased in loudness, but not in distinctness, by using a long instrument, or by connecting it with a hollow box, which answers the purpose of a sounding board.

I recommend the student a simple one, like fig. 2, well fitted to his ear, and he will need no other instrument; but he will need some perseverance and attention to learn the use of it, and to understand its indications.

THE

PATHOLOGY AND DIAGNOSIS,

&c. &c.

PART I.

PRINCIPLES OF DIAGNOSIS OF DISEASES OF THE CHEST.

A KNOWLEDGE of the condition of the chest and its organs, in health and in disease, is to be obtained by the study of those properties which are signs or indications of this condition. These properties are divided into two classes: the *physical properties*, which are those capable of directly impressing our senses; and the *vital phenomena*, or those which arise out of the vital endowments of the parts, and which, in combination with physical structure, constitute function. The physical properties are necessarily confined to the chest, its contents, and those parts mechanically related with it; but the vital properties of the chest and its organs affect, and may become evident in, various other parts of the body, through the connecting vital influences of the nervous and vascular systems. The latter, therefore, constitute an extensive and complex subject; and as the object of the present treatise is chiefly to explain how these properties become signs of health and disease, the physical properties, which are simple and intelligible, will be the first and chief subjects of consideration.

CHAP. I.

PHYSICAL EXAMINATION OF THE CHEST.

We examine the chest physically by those properties of form, size, proportions, relative position and density of its parts, at rest or in motion, which are within the reach of our senses. Thus we see and feel the shape and motions of the chest; we listen to the sounds which these motions cause, or which may be otherwise produced in the chest, as by the voice, percussion, &c., and we judge of the results of these examinations by comparing them with healthy standards. Now it is obvious that a good knowledge of the anatomy and physiology of the chest must greatly assist us in this examination; and this knowledge should be especially *topographic*, teaching where each organ lies and reaches in relation to the exterior, so that when we view, feel, and listen to the chest, we may be able to map out the general outlines of the organs within. This knowledge is to be obtained not only from the common study of anatomy, but also from the frequent comparison of the interior with the exterior, when bodies are opened in the dead-room of hospitals, and from the habit of physical examination itself.

I have been in the habit of illustrating this subject in my lectures by means of coloured diagrams, in which the contents of the chest are exhibited transparently by different tints of the figure. It is a much more difficult matter to show the same things in black and white; but I have made the attempt, and I hope that the student will find the accompanying figures assist him to understand the topography of the organs of the chest, and their relation to physical signs. They are an attempt to express more than the somewhat similar diagrams in Dr. Forbes' able article on exploration of the chest in the Cyclopedia; the outlines of organs being represented not only through the walls of the cavities, but also by fainter lines through superjacent viscera. Thus the liver (4), the heart and vessels (3), and the stomach (5), are seen through the thin lobes of the lungs. Fig. 3 is added to show the state of the chest on a full inspiration. One error should be corrected: the nipples in fig. 1 are somewhat too high; in the erect posture, with the arms hanging, they generally lie on the upper edge of the fifth rib.

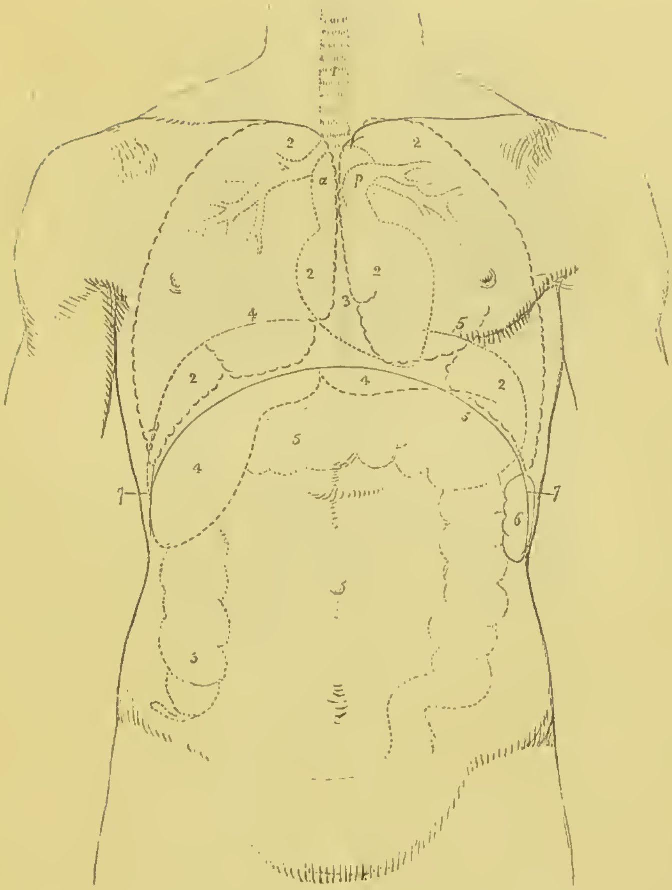


FIG. 1.—GIVING A TRANSPARENT FRONT VIEW OF THE FORM AND RELATIVE POSITION OF THE CONTENTS OF THE CHEST.

1. Trachea dividing into bronchi, which are marked in fine dots.
2. Lungs, bounded by line of short curves.
3. Heart and great arteries, bounded by fine dotted line. *a*, aorta; *p*, pulmonary artery.
4. Liver bounded by a dotted line, rising behind the lungs in the inner arch of the diaphragm.
5. Stomach and intestines in a dotted curved line, and rising in the inner arch of the diaphragm to 5 on the left side.
6. Spleen.
7. Front arch of the diaphragm.

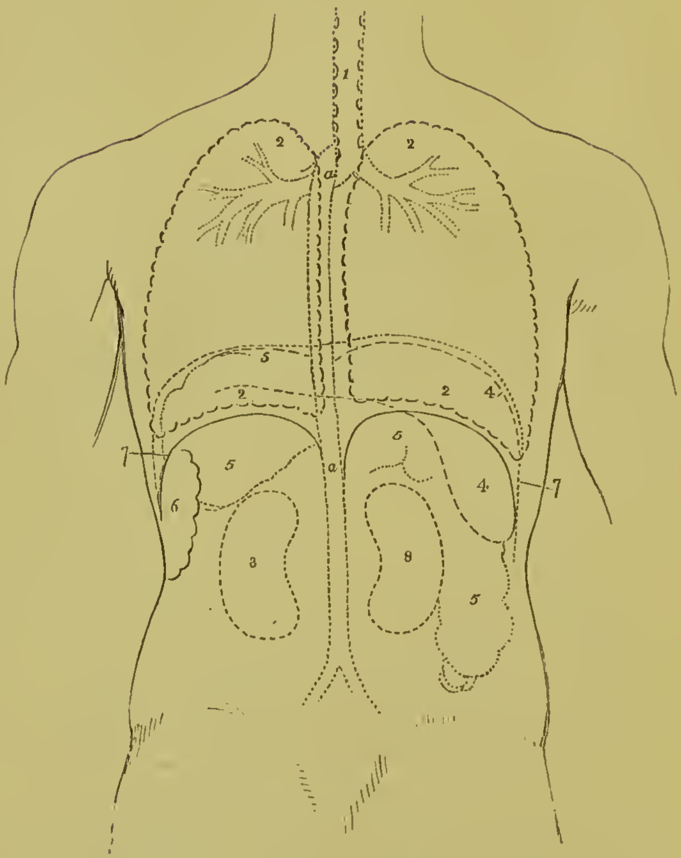


FIG. 2.—TRANSPARENT BACK VIEW OF THE CONTENTS OF THE CHEST. Numbered as in fig. 1, except 8. Kidneys. 7. Back arches of the diaphragm.

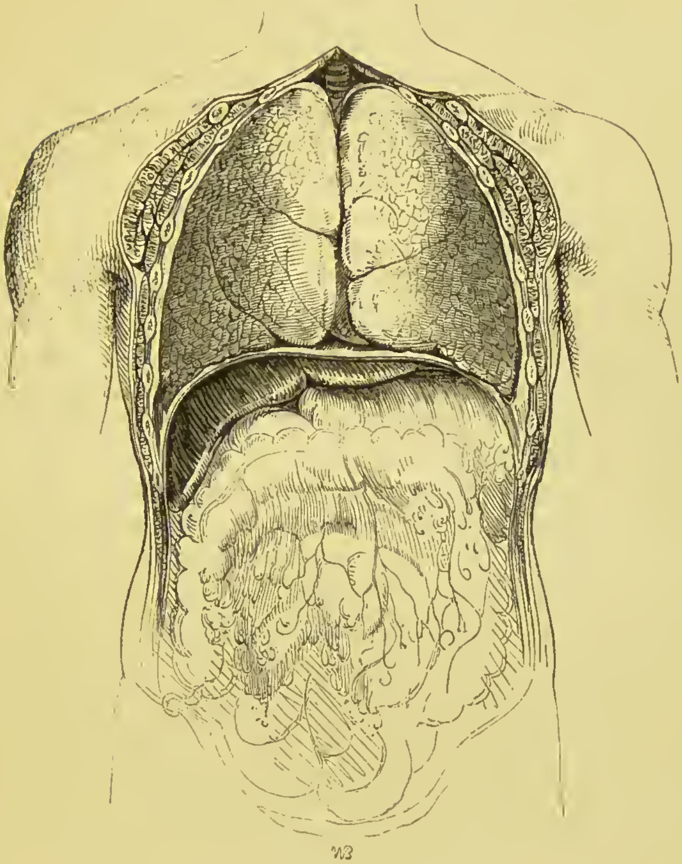


FIG. 3.—Shows the manner in which the lungs cover the heart, &c., on full inspiration.

Examination by sight and by touch.

The patient being in a standing, sitting, or lying posture, with his arms, legs, and trunk symmetrically placed, and the chest, if possible, entirely uncovered and exposed to a good light, we view it in front, behind, and from above, carefully marking its form and proportions, and the corresponding prominences and depressions of the two sides. Most healthy chests are nearly symmetrical, the two sides corresponding in shape and size. There is, however, in almost all instances, a slight superiority in the measured dimensions of the right side, and this is not unfrequently perceptible to the eye in the lower part of the chest, especially in the back. I have also remarked in a great many healthy chests, that the left side of the chest at the upper ribs in front is somewhat higher than the right.* These trifling exceptions to the symmetry of the chest correspond with physiological heteromorphisms described by M. Woillez to have occurred in more than a fourth of the healthy chests examined by him. The sternal depressions and prominences mentioned by the same observer sometimes to occur independently of disease, do not interfere with the symmetry of the chest; and spinal deviations which have this effect can be readily detected by an inspection of the back.

In spite of the occasional and even frequent occurrence of the anomalous formations above described, there is sufficient general regularity in the shape and motions of the chest to render the changes caused by disease available as signs.

I have said that the chest should be viewed from *above*, as well as from before and behind. This may be done when the patient is seated on a low seat, with the head a little inclined forward, and the observer stands behind or beside him, and looks down on his shoulders. A view is thus obtained of the depth of the chest from front to back; and in this way may often be detected between the two sides a want of correspondence that is not perceptible by the ordinary modes of inspection. If the patient's strength do not permit him to stand or sit, the chest may be inspected when he lies on his back by the observer standing at the foot or at the head of the bed, from which points the corresponding parts of the two sides can be best seen.

By inspection we judge not only of the statical condition of the chest, but of its motions likewise. Feeling by the hands will assist the sight; and when we inspect the chest, desiring the patient to breathe in various degrees, with our hands and eyes fixed on corresponding points of the two sides, we watch and feel the amount and equality of the motions. If the chest is healthy, the motions are as uniform as the chest is symmetrical. The clavicles, scapulæ, and upper ribs rise, the lower ribs rise and spread, and the abdomen swells as the diaphragm descends at each inspiration. By attentively watching and feeling the chest, we may often likewise so trace the limits of some of the movements, as to indicate the boundaries of the chest. Thus the lower ribs are pressed outwards by the displacement of the abdominal viscera at each descent of the diaphragm, and cause a prominence below the limits to which the lungs descend, with a slight flatness or hollow above. The appearances which I have thus explained were first pointed out by Dr. Edwin Harrison, as visible indications of the height at which the liver and the stomach lie in contact with the walls of the chest. In tall or slender persons there is often another slight depression two or three inches higher up, corresponding with the upper part of the vault of the diaphragm, which is overlapped by the lower margins of the lungs. These marks, together with the situation of the depression at the top of the ensiform cartilage, and of the beat of the apex of the heart, will sometimes enable one to discern the precise boundaries of the thoracic and abdominal cavities, and therefore assist to distinguish between the diseases of their respective viscera.

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* The fuller developement of the right side was ascribed by Laennec to a law of conformation, traceable through the whole animal creation. Others, again, have attributed it to the habitually greater exercise of the arm on this side, and it is said that left-handed persons present the preponderance on the left side. Further observation is wanted to determine this point; but as far as my own goes, it would lead me to regard the exercise of the arm as a cause of increase only in the muscles on the chest, and not in the chest itself. So, too, M. Woillez found that persons employed in trades requiring much use of the upper extremities presented a smaller average capacity of chest than usual. I apprehend the true cause of the fuller size of the lower part of the right side and upper front of the left, lies in the resistance presented by the liver on the one side and the heart on the other. This resistance directs the forces of respiration more strongly to the adjacent outer walls. This view accords well with the fact observed by Woillez and others, that the same parts of the chest are the most ready to enlarge under the influence of disease.

The intercostal spaces and the hollows above the clavicles are also fit marks for comparison between the two sides. They are strongly marked during full inspiration, and are so more than usual when the entry of air into the lungs is difficult from obstruction of the tubes; and less than usual when the obstruction is more in the tissue of the lung from internal effusion or external pressure.

Applying the hand on the region of the heart, we feel the relation of the respiratory motions to that organ. After a full expiration, the heart is felt beating at the cartilages of the third and fourth ribs and under the sternum, as well as at the fifth rib; but as the ribs rise and the lungs expand by inspiration, we gradually lose the beating, and at the completion of a full inspiration, if it be felt at all, the beat will be as low as the sixth rib. It would be tedious to dwell longer on the details of the healthy appearances and motions of the chest, but the student should lose no opportunity of familiarising himself with them.

There are some general varieties of disordered respiration which may be distinguished by watching and feeling the motions of the chest. Healthy or perfect respiration is diaphragmatic and costal; but disease may limit the motions to the ribs or to the diaphragm. Thus when the diaphragm is prevented from descending by acute pain in it or below it, or by pressure from below, the respiration is wholly performed by the raising of the ribs, and is called *heaving*, *thoracic*, or *costal*. When, on the other hand, the ribs are immovable in consequence of pain, ossification of the cartilages and ligaments, or paralysis of the intercostal muscles, the breathing is wholly *diaphragmatic* or *abdominal*.

When one side of the chest moves much less than the other, or when a part of one side moves imperfectly, the respiration is *partial;* and this partiality of movement may have its cause in the walls of the chest, or, as more usually, it may proceed from impermeability of the corresponding portions of the lung, in consequence of various diseases. Thus when lymph or tuberculous matter in the tissue of the lung, an obstruction of the bronchi, an effusion into, or a contracted adhesion of, the pleura, prevents the inflation and collapse of a part of the lung, the corresponding walls of the chest will be resisted in their motions, and will be fixed in proportion. Thus in phthisical patients we sometimes see the ribs below the clavicles sunk on one side, and scarcely moving in respiration; in pneumonia and pleurisy the lower ribs are more commonly fixed. It is proper to mark further how they are fixed: whether in a state of dilatation, or in one of collapse; whether the affected part remains full after expiration, or is still sunk after inspiration; or whether it is fixed in an intermediate state. We may thus, in some instances, distinguish between the different causes of obstruction. In this and other cases, however, tact and vision are useful as general means of survey, preparatory to other more accurate modes of examination.

Mensuration of the chest is a more exact method of detecting inequalities between the two sides. It is generally practised by fixing with the finger the end of a piece of tape or string, at the mesial line of the sternum, and passing the tape horizontally around the chest to meet at the same spot; then by taking it off at the point where it crosses the spinous process of the dorsal vertebra, the measure of the two sides may be at once compared. Great care must be taken to pass the tape horizontally around corresponding parts, and attention should also be paid to the degrees of the respiratory act. The most accurate mode is to compare the measurements of the two sides on a full inspiration and expiration, as well as in the intermediate state; or without the troublesome process of removing and measuring the tape, it may be sufficient to remark how it is tightened and slackened on the two sides by the motions of respiration. Dr. Stokes recommends the use of graduated callipers to measure the depth and height of the chest, as well as its circumference, which alone is given by the tape. Such an instrument would doubtless give more exact results; but as it is not likely to be generally used, I may mention that I have been in the habit of using the tape for the height also, by measuring from the bottom of the sternum, or from the umbilicus, to the hollow under the humeral end of the clavicle; from the latter spot to the spinous process of one of the lower dorsal vertebræ; and from the top of the sternum to the lowest point of the margin of the ribs at the side. These expedients, together with the practice of inspection downwards on the shoulders for the antero-posterior diameter of the two sides, are generally sufficient to furnish the comparative dimensions of the sides of the chest.

Besides external measurement, which is essentially *comparative between the two sides*, there have been various attempts to measure the internal capacity of the chest, by noting the quantity of air that can be exhaled or inhaled at a breath into or from a glass jar over water. The great objection to these as means of diagnosis is, that they depend on the strength of the patient as much as on the capacity of the chest; and a weak nervous person with sound lungs thus tested would rank lower than a pleuritic or phthisical patient, whose muscular energies are still considerable.

So much for the modes of examining the chest by sight, touch, and measurement. They may often discover to us extensive disease, but they will not inform us of its nature, and they cannot detect the slighter degrees and forms of disease. The chest may be motionless, distended, or contracted, in parts; but whether from diseased lung, obstructed air-tubes, liquid or air in the pleura, or other cause, sight and touch will rarely inform us.

Examination of the chest by hearing.

We are led then to pursue the examination by another sense, which may reach beyond the surface,—the sense of *hearing*. This is not so habitually cultivated as our sense of vision. "Seeing is believing:" to *see a thing* is synonymous with to understand it. The same remark is not applicable to hearing; yet in cases where we have much exercised this sense, its indications become nearly as instructive as those of sight. Thus we know the different sounds of carriages and horses in the street, the footsteps of walkers, the noises of rain falling, wind whistling, of hammering of iron, wood, or stone; and these several noises are the acoustic signs of the nature or physical character of the various bodies that produce them. So also the acoustic properties of the chest and its organs; the sounds that are produced in them, or elicited from them, may, when studied, instruct us as to the nature and condition of the parts in which they are produced.

The acoustic phenomena of the chest should be studied not only by mere experience, like that by which an infant gradually becomes acquainted with the objects around it, but also more rationally, by a generalisation of such experience, in the laws according to which the phenomena occur. We must accustom our ears to the sounds in all their varieties, that we may be able by experience to know and to distinguish them; but to understand their import, and to read the interpretation which they give of the condition of the parts which produces them, we should study them through the laws under which they occur. We must consider what sound is, how it may be produced, transmitted, and modified; how the contents of the chest may produce it, and when produced can change it; and by comparing its general properties with the mechanism of the chest and its organs, we shall be prepared to understand and arrange the phenomena that experience has discovered, or may hereafter reveal to us. By thus learning the acoustic relations of the chest, not merely as isolated facts, but as parts of an applied science, we may be enabled to escape many of the errors into which unintelligible matters of memory might continually lead us, and we shall be acquiring a rational pathology instead of resting on an empirical diagnosis.

On the nature and properties of sound.

A few considerations relating to sound in general will enable the student pretty well to understand the acoustic phenomena of the chest; and although he may be referred to the best modern works on natural philosophy for a fuller illustration of the subject, I shall here endeavour in a few words to divest it of some of its technicalities, and to explain simply those properties of sound which are concerned in producing signs of health or disease.

Sound is not a matter or a separate thing; it is matter in motion of a particular kind, and this motion being communicated to the matter in contact with our organs of hearing, produces that sensation which we designate as that of sound. The same motion may often be seen and felt by sight and touch, as in the vibration of a bass cord; and when a person speaks, the motion of the voice may often be felt in some parts of the neck and chest. But our organs of hearing are more sensitive of this motion than our eyes or fingers: they can feel finer degrees of it, because they are specially adapted to it.

If we inquire what is the kind of motion which constitutes sound, we shall find that it is resisted motion, or *motion resisted with a certain force.* The moving force and the resisting force, impelling the matter alternately to and fro in opposite ways, constitute what are called the *vibrations* of sound. These may be seen in a vibrating cord, and may be illustrated by the analogous but slower motions of a vibrating pendulum.

The *transmission* or *conduction* of sound is the communication of the sonorous motion from one body to another, as one ball striking another ball moves it. The *reflection* of sound is the refusal or rejection backwards of this motion by bodies which cannot receive it, as a wall throws back the motion of a ball. Sound is most readily *produced* and sustained in bodies of uniform density and elasticity, the particles of which transmit and continue, and do not reject or choke, each other's motions. Hence tense and rigid bodies, like metal, wood, and glass, produce and conduct sound better than those which are flaccid and soft, such as cloth, clay, membrane, &c. Bodies of very different density and elasticity do not readily receive sound from each other; because their powers of motion differ in force and extent, and cause them to reflect or to choke each other's motions. Thus a sound produced by air is intercepted by a solid; and that produced by a solid is transmitted by a solid of the same density and elasticity far better than by air. Bodies varying in density, such as soft, spongy, or porous bodies, are the worst conductors of sound, because the unequal elasticity of their parts chokes the motions of sound. The sound of a body much more dense than air, such as metal, may be communicated with greater freedom to air by the medium of a third body of intermediate density, such as wood; and this effect may be increased by extending the surface, and lightening the mass of the third body, as it is done in the sounding boards of musical instruments.

The *note* or *pitch* of a sound depends on the frequency of the vibrations, those of the highest or shrillest notes being the most frequent. The *duration* of a sound depends on the continuance of the vibrating motion, and this will be determined by the amount and uniformity of the elasticity of the body. Thus glass and metal are highly and uniformly elastic, and are therefore, when hung free from other bodies, capable of giving prolonged and musical notes. Soft bodies, on the other hand, such as cloth, flesh, &c., which are inelastic and not uniform in density, give only the shortest and slightest sounds, their vibrations being stopped by the yielding of their particles, or by their irregular motions, which, like those of pendula of different lengths, soon clash with and stop each other. In a similar way these soft bodies muffle or choke the vibrations of sonorous bodies with which they may be brought into contact. Again, solids which are rigid, yet variable in density, like wood, are capable of producing loud sounds, but not of prolonging them, and they prove excellent conductors of sounds produced in other and more uniform solids. The *body* and *loudness* of a sound depend on the extent and force of the vibrations which reach the ear.

The *sources* of sound are such impulses as affect bodies suddenly, or with some force, so that there be both motion and resistance. Thus the percussion, collision, friction, tightening, and breaking of solids cause sound. Sounds are not often produced in air or in liquids, but by the aid of solids, which either communicate the motion or offer the resistance. Thus we hear the wind only when it whistles in the keyhole, in the rigging of a ship, the leaves of a tree, or the like; and the sounds of wind instruments depend on the motions of a solid, as in the case of the hautboy and clarionet; or on the resistance of a solid, as in the case of the whistle and the flute. Liquids and air together will also readily generate sound by their impulses against each other, and thus are caused the various bubbling and rushing noises of liquids, from the frothing of beer to the roar of a cataract.

Examination of the chest by percussion.

As the nature of metal, wood, and other bodies is tested by the sounds which they yield on being struck, so we strike or tap on the chest to judge of the nature or condition of its materials. The practice of percussion as a mode of diagnosis we owe to Avenbrugger: it has been applied and improved by Corvisart, Laennec, Piorry, and others, and now constitutes an important mean of diagnosis in diseases of the chest and abdomen. When the principles on which its indications depend are well understood, the practice becomes easier as well as more instructive; and as I believe that no preceding writers have exposed these principles correctly, it will be well, after stating the general phenomena of percussion, to explain them by the aid of a few easy experiments.

The chest, when struck abruptly with the ends of the fingers, yields a rather deep and not very short sound; which implies that the vibrations are not quick, and that they do not instantly cease. If we strike in the same manner on the thigh, a very different sound results—a short dull tap, implying that the vibrations have no strength or continuance. The same dead tap is obtained on striking the lower part of the chest on the right side, where the liver lies; but all those parts which cover the lungs yield more or less of the deep hollow sound. How are the vibrations which constitute this sound in the air contained in the lungs or in the solids of the chest? To answer this question, we must refer to the known properties of hollow bodies with regard to sound. Thus if we take an indian rubber bottle and strike it, we find that its note, which depends on the vibrations of the air within it, is quite different when its mouth is opened, from that which it yields when it is closed. But closing the glottis, which is the opening into the hollow of the chest, does not materially change the sound of pectoral percussion. Again, the sound of hollow bodies is deep in proportion to their size. Thus a large indian rubber bottle gives a much deeper tone than a small one; and its note is raised on diminishing its cavity by compression. It is not so with the chest, for enlarging or diminishing its hollow by inspiration or expiration does not in this way change its sound on percussion; the extremes of these actions only slightly raise it in another way. As, then, the sound of percussion of the chest does not follow the law which regulates sounds produced in the air of hollow bodies, we must conclude that it is seated in the solids; and if we study the construction of the chest, we shall see how well its solids are adapted to vibrate. Composed of layers of membrane, thin muscle, and integument, stretched on an elastic frame of bone and cartilage, the walls of the chest are free to vibrate, so long as the organs within do not check their motions. Were there nothing but air within, these motions would be perfectly unembarrassed, and the sound would be more prolonged and hollow in consequence; deriving also an additional hollow tone from the note of the cavity within; as we find it exemplified in pneumothorax, or with an inflated stomach in the left side of the chest. Again, the light, soft, spongy tissue of the lung scarcely interferes with the free vibrations of the walls, whilst the slight sound of its own which it yields is equally deep with that of the walls with which it becomes combined.

Hence, where healthy lungs lie in contact with them, the walls of the chest give a deep clear sound. But below the sixth or seventh rib on the right side, which is over the liver, and to the left of the lower part of the sternum, which is over the heart, the sound is dull and short, the vibrations being checked by these solid organs beneath; so it is obvious that morbid changes of the organs, such as a condensation of the lung, or serum effused in the pleural sac, would in a singular way arrest the vibrations, and render the sound of the chest dull in those parts where these changes occur. On the other hand, changes of an opposite kind, such as dilatation of the air-cells of the lungs, or air in the pleural sac, may make the vibrations of the walls more free than usual, and thus increase the sound obtained on percussion.

These illustrations are enough to show the general principles of the acoustic examination of the chest by percussion. It is a test of the density and elasticity of the materials within the chest; as diseases alter these qualities, so will they alter the sound on percussion, which may thus announce their presence. A few more considerations will suggest some practical application of these principles.

As we have seen that the walls of the chest give the sound which we hear on striking the chest, so it is plain that they must be sufficiently tense and elastic to vibrate on being struck. The chests of some persons are so loosely put together and so flaccid, that they give but little sound, although the organs within are quite healthy. In others, again, there is such a mass of fat and loose integument on the chest, that the walls are completely muffled by it, and they sound but little on percussion. The same difficulty occurs in other cases in certain regions where muscles of considerable thickness, or the mammæ in females, lie on the walls. In other instances, again, the walls of the chest are so drawn in by contracted adhesions of the pleura, that they are too tight to vibrate, and give a hard or dull sound, although the lungs within them may be comparatively healthy. In all these cases we must give to the part struck the equal tension which is wanting, by pressing on it a small piece of some firmly elastic body, such as wood, ivory, whalebone, stiff india rubber, or some such substance. This when struck gives sound enough; and if it be firmly applied to the chest, the density of the contents within will modify this sound, just as it modifies that of percussion on the naked walls of the chest. The sound obtained by striking a little plate of ivory or wood thus pressed on the chest, is the same in character as that of striking the chest itself; but it is louder, and as percussion on it gives no pain, the stroke can be applied with such force as to make the vibrations reach the interior through any thickness of fat or muscle. By these means, we can test the sonorous qualities of the thoracic viscera through the scapulæ and muscles of the back, and through fat or œdematous integuments of any thickness. In this way, too, we can try the resonance or sonorous quality of any part of the abdomen.

We owe this method of *mediate* percussion to M. Piorry, who calls this percussion-plate a *pleximeter*. Mediate percussion is so much better than the immediate kind, that it is now generally preferred. There is, however, an improvement on it, which was, I believe, first proposed by Dr. Skerrett; it is to substitute for a pleximeter the fingers of the left hand. This mode of percussion has the advantage of convenience as well as of yielding distinct results. Its adaptations are soon found out by a little experience; in fitting the fingers to the inequalities of the chest, sometimes single, sometimes together, sometimes with their palmar surface outwards; but generally with this applied to the chest, and the back to strike on, with other varieties of manipulation to be hereafter noticed.

To understand the varieties of sound of percussion, (or, as it may be more briefly called, *stroke-sound*,) and their situations in the chest, it is necessary to bear in mind how the contents of the chest lie in relation to the surface. This varies considerably in different healthy individuals; but the following may be given as an average statement of the position of the thoracic organs after an ordinary expiration.* The lungs are in contact with all the upper and middle portions of the walls of the chest. On the right side they reach down to about the sixth rib in front, and the eighth rib at the side, below which the liver comes in contact with the walls: the lungs reach still lower in the back. On the left side they reach to about the seventh rib in front, except within two or three inches of the sternum, where they do not

* The figures already given will aid the student in following and understanding this description.

reach lower than the sixth rib, there being a space of from one to two superficial inches under and to the left of the sternum, where the heart lies in contact with the walls; at the side they reach to the eighth rib, whereabout they are bounded by the stomach and spleen, which, with the colon also, bounds them behind, where they reach a little lower. Inspiration greatly alters these limits, both by raising the ribs and expanding downwards and outwards the lungs, which then reach a rib lower, and a full inspiration may bring them in contact with nearly the whole of the thoracic walls,(as seen in fig. 3,) while expiration has the converse effect.

The sound on percussion corresponds with this description, and by it we may therefore know the position of the organs in a living subject. Thus in all the upper parts of the chest before, behind, and at the sides, the stroke sound is clear, and equal in both sides. There is also some clear pulmonary sound in the inferior parts of the chest down to the limits to which the lungs reach. But below the fourth rib in front on either side, (figs. 1, 4 and 5,) although the lungs are in contact with the walls of the chest, their lobes are not thick, and beneath them lie the liver on the right side and in front, and the heart and stomach on the left front. The vicinity of these organs modifies the stroke-sound; and the more so the nearer they approach to the surface, where the lungs become thin towards their margins, until they quite give place to the peculiar sounds of these respective viscera about the limits before named. There is therefore a very slight deadening of the pulmonary sound below the fourth rib on the right side, below the middle of the sternum, and below the third rib near the sternum on the left, and this deadening increases down to the margin of the lungs, where the sound has the perfect dulness of the solids of the liver and heart. Further to the left, the sound takes more or less of the hollow tympanitic character of the air-filled stomach. It appears, then, that the stroke of percussion reaches to a considerable depth; to organs an inch or more from the walls: and whatever it reaches may modify the sound. This suggests to us, that by varying the force of the stroke, we may make the impulse of percussion reach to different depths, and derive the character of its sound from the superficial, or from the deep-seated organs, as we will. Thus where the lung overlaps the liver, (below the upper 4, fig. 1,) *strong* percussion will give a shorter, deader sound than gentle percussion. Strong percussion receives the character of its stroke from the liver as well as from the lung; whilst gentle percussion, such as by filliping with the finger and thumb, does not pass through the thin layer of lung, and gives still the pulmonary sound. The same mode of percussion may distinguish the utmost limits of the lungs over the heart. It is more difficult to determine by percussion the precise limits of the lung on the left side; for in consequence of the loudness of the hollow stomach sound, and the facility with which it may be elicited, it is apt to disguise the pulmonary sound even with the most gentle percussion. In this case more may be done by observing the amount of expansion of these parts by a full inspiration. The extent of this stomach sound varies according to the state of gaseous distension of the stomach: it not unfrequently reaches in a slight degree above the mammilla, and nearly to the axilla. There is sometimes a slight dulness on the left side behind, corresponding with the position of the spleen; and in case of enlargement of this organ the dulness may become extensive.

It must not be forgotten that the motions of respiration may produce changes in the character and position of some of these stroke-sounds. Inspiration, as it enlarges the lung, renders the pulmonary sound clearer, and extends it over every part of the heart, and over a considerable portion of the liver. As the complete and equal enlargement and contraction of the chest, as seen and felt, are signs of the free conditions of the respiratory organs, so the stroke-sound becomes an additional sign of the healthy action, in proportion as the clear pulmonary sound is extended at each expansion of the chest. Percussion is a test, therefore, not only of the statical condition of the lungs, but of their dynamical state also. This point is not enough attended to by auscultators; yet the neglect of it not only would deprive us of additional signs, but would tend to render deceptive the results of statical percussion. For example, in judging of the goodness of sounds of percussion, we generally compare the sounds on the two sides of the chest, where in health the structures and sounds are the same; but if we do not attend to the movements of respiration, we may strike one part when the chest is contracted, and the other when it is full, and obtain results which differ from this cause only, and not from any internal change. In practising comparative percussion, therefore, in cases requiring delicacy, it is proper to desire the patient to hold his breath for an instant while the comparison is made, and it is often useful to try the sounds when the chest is expanded to the utmost, when it is contracted, and in the intermediate states.

The varieties in the stroke-sound from special diseases will be considered when those diseases are described, but a few examples will be useful here to illustrate the subject. In phthisis, the indurations of the upper lobes of the lung are often small, and so scattered through its substance, that they scarcely affect the stroke-sound ; but by a full expiration they are brought closer together, and if more on one side than on the other, they might then sensibly deaden the sound on that side, especially if gentle percussion be used below the clavicles, and not on a very small surface. Again, the indurations, especially if of some standing, tend to restrain the lung from its full expansion; and if there be a difference on the two sides, it thus may be detected only on a full inspiration. In the disease called emphysema of the lungs, the air-cells are permanently dilated ; they contain an unusual quantity of air, which expiration cannot expel ; this may be detected by percussion used as a dynamical test ; the regions of the heart and middle part of the liver being covered by the permanently distended lung, give, even after expiration, a clear sound. There is one more point to be noticed respecting percussion at the extremes of the respiratory act. Full inspiration makes the sound clearer ; full expiration has the contrary effect ; but they both raise the note a little ; they make its pitch higher. This is because forcible inspiration and expiration are muscular actions which strain the walls of the chest, and render the vibrations quicker, and therefore the sound higher, as in the tightening of a drum.

It is unnecessary to add more on the principle of percussion ; but it will be useful to give some directions with regard to the practice. In this as in every other art requiring some manual dexterity, and the exercise of the senses, practice is necessary to familiarise the beginner with the phenomena and the mode of obtaining them. There is no better way for the student to get this familiarity, than by percussing his own chest. A few minutes before dressing in the morning, spent occasionally in studying before a looking-glass the outward marks and different sounds of the regions, will go far to instruct him in the practice of percussion, and in the character of the sounds in health ; and the awkwardness and annoyance inseparable from first attempts will be felt only by the person who is least likely to complain of them.

In obtaining the sounds of percussion, the student soon finds that some dexterity is necessary even in mediate percussion, which is the easiest mode. The fingers or pleximeter should be closely pressed on the walls of the chest, and if the object be comparison between the two sides, they should be placed on corresponding parts, whether between the ribs, along them, or across them. Care should also be taken that the mode of striking be the same, whether it be with one or several fingers, with their tops, (in which case the nails should be kept short,) or the flat of the last phalanx, or with the knuckles, each of which modes is sometimes preferable. Filliping with the middle finger and thumb often gives more uniform and delicate results; especially when the patient is in an inconvenient position, or suffers from tenderness of the walls of the chest. This is also the best mode for abdominal percussion. When striking the clavicle, attention should be paid to what part is struck; for the sternal portion of this bone always sounds much clearer than the humeral end. So also, in percussing this or any other part, the direction of the stroke should be perpendicular towards the lungs, and not sideways, or the sound will be modified, not by the lungs, but by the adjacent muscles or other parts towards which the impulse is directed. It is from neglecting this precaution, that beginners sometimes get nothing but dull sounds all over the chest. It is not generally necessary to use much force in percussion; in fact, many of the most valuable results are obtained by gentle mediate percussion; but the mode must be varied in different cases. When it is desired to test the density of a small spot, percussion with a single finger is best; whilst, for trying a surface of greater extent, flat percussion with several fingers answers better. In doubtful cases, it is proper to try both. In percussing the regions of the back and shoulders, the bony prominences of the scapulæ and ribs should be sought; for these transmit the impulse to the interior far better than the thick layers of muscle. But as the scapulæ are moveable bones, it is necessary to see that they are in corresponding places on both sides; and to ensure this, and to increase the tension of the muscles, it is well, in examining these regions, to desire the patient to cross his arms in front, and to bend his head forwards. At that part of the chest near the humeral end of the clavicle—a most important region for examination, there is often a falling away of the chest, and the more tense the pectoral muscles are made, the further they are removed from the walls. Here, then, instead of making the muscles tense, they must be relaxed, by letting the elbow hang close to the side, whilst mediate percussion is practised in this region.

The best posture of the patient for percussion is erect or sitting; and in comparing the two sides both before and behind, the observer should be directly opposite the front or the back. When the patient is lying down, the sound is modified by the matter on which he is lying; if it be a soft feather-bed, the sound is more dull; if a mattrass or anything hard, the sound will often be increased, because the elasticity of the contents of the chest is increased by the unyielding matter behind it. So also the vicinity of a wall or other hard surface causes a reverberation, which gives to the side nearest to it too loud a sound. As most of the effects of percussion are judged by comparison, the chief object of the cautions which I have given is to take care that any of the external causes of modification may not act unequally on the different parts of the chest. The patient may sometimes be removed from the influence of these to the middle of the room; and when this cannot be done, these modifying causes must be equalised as much as possible.

I need say little about pleximeters, for they are not generally necessary. M. Piorry much exaggerates the advantages derived from them. A thin circular plate of wood or ivory, with two projections or a raised rim by which it may be held firmly to the chest, is one of the best forms; the surface to be struck should be covered with soft leather, to prevent the clack of the fingers on it. I have often used a little oval piece of boxwood, about an inch long, three-quarters of an inch wide, and one-eighth of an inch thick, with a strong handle two inches long, rising from its outer margin at an angle of about forty-five degrees. The handle is convenient for holding it firm to the chest without interfering with the percussing fingers. The chief precaution necessary in using it, is to take care that it be applied flat, and not tilted. A simpler and perhaps a still better instrument is a flat slip of pretty stiff whalebone, three or four inches long, and half or three-quarters of an inch broad, slightly bent at its middle. Half of this, covered with soft thick leather, fits well as a percussion plate to any part of the body, and, held by the other end, the elasticity of the material secures its closer apposition.

Auscultation of the respiration.

Let us now inquire into the modes of producing sounds in the chest which may prove signs of the condition of the organs within. The contractions and relaxations of the muscles of respiration are in general too gentle to cause sound; but, when forcible or sudden, they sometimes produce a sound of tightening, a kind of muscular sound. This is often evident when there is an abrupt catch in the breathing, and during the act of coughing, though it does not furnish any sign of importance. But the internal motion and passage of air to and from the lungs produce sounds; and inasmuch as this passage of air is the great object of respiration, we might expect to find in these *breath-sounds*, signs of the manner in which this object is accomplished. These and most of the other acoustic motions of the chest were chiefly discovered and described by Laennec, who may well be regarded as the father of the art of auscultation. I proceed to describe and to explain them consistently with physical laws and the observed characters of the phenomena.

The air enters the lungs by atmospheric pressure, to fill the increased space made in the chest by the action of the muscles of inspiration. On its way to the most expansible parts of the lungs, the fine tubes and cells, it strikes against the sides and angles of the larynx, trachea, and its ramifications, with force sufficient to produce a particular hollow blowing sound. We may hear this on applying the ear to the forepart of the neck, or to the top of the sternum. As the current of air becomes subdivided, and spread in the small bronchi, it loses a part of its velocity, and the sound becomes of a more diffused and less hollow character; it is more like the sighing of a gentle breeze among the leaves of trees, and, in passing into the cellular terminations, all of the hollow tubular sound is lost, as may be perceived on applying the ear to most parts of the chest where the lungs lie. This sound seems to depend here chiefly on the impulse of the air against the angles and sides of the minute tubes and cells, but partly also on the opening and stretching of these cells, and perhaps partly on a propagation of the louder sound of the passage of air in the larger tubes.* Where inspiration ceases, expiration begins; and a portion of air is pressed out of the cells and small tubes by the collapse of the walls of the chest, and by the contracting properties of the pulmonary tissues. There is a remarkable difference between inspiration and expiration: in inspiration air is the moving body, and, rushing through the tubes, distends the passive lung. In expiration, the lung is the moving body, and by its contraction (backed by external pressure) drives before it the passive air. In either case there is a pressure exerted between the air and the interior of the cells, and doubtless this proves the means of assisting the chemical changes that take place. But it is plain that there must be a difference between the sounds of inspiration and expiration. In inspiration, air moving with some velocity meets with the resistance of the angles and sides of the tubes and cells which it has to dilate, and this more in proportion to the suddenness and force of inspiration. Here must be sound in the whole passages of the air, from the nostrils down to the pulmonary cells. In expiration, the motion begins with the lungs, and the air passively yielding to it, there is not motion or resistance enough to produce sound, until, by the converging together of the small tubes, the impelled air is gathered into a current in the larger tubes, where, impinging against their sides with its now acquired velocity, it at length produces sound. These remarks explain why, in natural inspiration, there are three kinds of sound produced by the motion of the air through different parts, and hence called *tracheal*, *bronchial*, and *vesicular*. In expiration the bronchial and tracheal are heard only in certain situations, and the vesicular sound being obscure, there is little sound of expiration in most parts of a healthy chest. These differences, which were overlooked by Laennec, have been noticed by Andral, Louis, and Dr. Cowan, who ascribe their discovery to the late Dr. Jackson, a young American who studied at Paris. They deserve attention, and seem to be dependent on the causes which I have mentioned.

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* I do not think it advisable to perplex the student with the very different explanation of the sounds of respiration proposed by M. Beau, and ingeniously advocated by Dr. Spittal; (Ed. Med. and Surg. Journ. p. 138;) because I am convinced that it is erroneous. These writers consider that all the respiratory sounds are wholly generated in the larynx, and upper parts of the respiratory passages, and are merely propagated into the bronchi and lungs, deriving their various characters from different degrees of dispersion or diffusion of the sound. Of the many arguments which may be opposed to this view, I will only state the following. If the comparatively weak sound of laryngeal respiration penetrates so completely into every part of the lungs as to cause the vesicular murmur, how is it that the much stronger sound of the voice does not reach many parts of the periphery of the lung? How is it that in disease we find vesicular respiration so much exaggerated in parts as to become as loud as the tracheal, which has sustained no proportional increase? And how is it, that when a laryngeal rhonchus supplants the ordinary laryngeal blowing, we still have the vesicular murmur with the addition of the distant laryngeal rhonchus? The latter fact proves, indeed, that certain strong or sharp sounds in the larynx may reach all parts of the vesicular structure, and such were the sounds generated by Dr. Spittal with his air-condenser; but such sounds are very different from the hollow blowing of ordinary tracheal respiration. The ear recognises at once the identity of the former in the lung and at its source; but the sounds of natural respiration appear to be, as they for the most part truly are, produced under the spot where they are heard.

The sounds of respiration, or, as I would call them, *breath-sounds*, can be heard on applying the ear to different parts of the chest, being transmitted with sufficient distinctness through the parietes to the parts beneath : and as the healthy sounds vary in these different parts, we may judge of the natural distribution of the tubes by listening to these sounds. Thus we find, in any part of the neck at the upper part of the sternum, there is the hollow blowing sound which results from the passage of air to and fro in the trachea, which is therefore called *tracheal* respiration, which is loudest in expiration, and is then often audible to the unapplied ear. A little lower down than this, and over the space of from one to two inches on each side of the top of the sternum, between the scapulæ, and sometimes in the axillæ, there is generally more or less of the sound called *bronchial* respiration, its more whiffing or tubular character denoting that it is partly produced by the passage of air in the bronchial tubes, and this part of it accompanying expiration as well as inspiration. In most other parts of the chest is heard the pure vesicular respiration, which is a diffused equal murmur, caused by the air penetrating through the minutest tubes, and into their numerous vesicles or cells. This is chiefly confined to inspiration ; in many persons entirely so ; in others there is a short faint sound of expiration, which I suspect to be chiefly transmitted from the upper parts of the respiratory passages, on which the expired air strikes with most force.*

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* M. Fournet, a recent French writer who has produced a bulky volume on auscultation, in which he shows more talent for refinement than for useful improvement or originality, attempts to represent the comparative duration of inspiration and expiration in health by the figures 10 and 2. Although such numerical expressions may be convenient for brevity's sake, I would warn the student

The question naturally occurs, why is the bronchial respiration heard in comparatively few parts of the chest, when bronchial tubes of considerable size are distributed in so many different parts of the lungs, within an inch or less of the surface? Why is not the sound a mixture of the tubular and vesicular sounds? The answer and explanation is, that in consequence of its softness and inequality of density, the healthy tissue of the lung is a bad conductor of sound, and does not transmit the sound of bronchial respiration to the surface, except in points where the tubes are large, and approach quite close the walls of the chest, and even here it is mixed with vesicular sound. The flaccid tissue, composed of the different materials, membrane and air, effectually arrests all the slighter sounds produced in the tubes within it. An important corollary from this is, that as this arrest of the sounds of the interior depends on the light spongy structure of the lung, so any disease increasing the density of that structure augments its conducting power, and enables it to transmit the sounds. Hence we find that a great increment of solid or liquid in the lung, as in pneumonia or tuberculous disease, or the compression of its superficial parts by a moderate quantity of liquid in the pleura, as in a recent pleurisy, often not only diminishes the vesicular murmur in consequence of the obstructed state of the cells, but also adds a bronchial or tubular sound of expiration and inspiration in those parts where naturally the sound is purely vesicular, and confined to inspiration. Such morbid sound is therefore much sharper and more distinctly tubular than the natural bronchial respiration described above, which is always mixed and obscured with the sound of the vesicular expansion.

As the several sounds of respiration depend on the resisted motion of the air, so they vary according to the velocity of that motion, and to the degree and nature of the resistance to it: they are loud when the air passes in and out forcibly and quickly, and low when it passes gently and slowly. So, on listening to a person's breathing, it may be scarcely audible at its ordinary rate; but if he breathe quick and short, it will be distinct enough. Taking a long breath may not answer the same purpose, for although much air is thus taken

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against their too definite character: numbers here can only be approximative; and it would be safer to say that the sound of expiration, when audible at all in health, has not more than a fourth or fifth of the duration of the sound of inspiration.

in, it may not enter with sufficient rapidity to cause the increased sound. Coughing answers better, for the full inspiration which succeeds coughing is generally quick also ; and it is often useful, where the sounds are obscure, to magnify them by this more forcible act. But there is a limit to this power of increasing the sound of respiration by increased effort. If an individual tries to breathe very hard and quick, as after violent exertion, the movements of the lungs cannot keep pace with those of the external muscles of respiration, and the air does not freely enter ; the sound will be diminished or altered, rather than increased.

As we can vary the sound of respiration by varying the act in the same individual, so we find that a difference exists naturally in different individuals : in some, as in many robust adults, the ordinary breath-sound is very low and faint ; in others, as in children, in nervous females, and in slight irritable persons, it is loud and distinct. In the last-mentioned cases, the respiratory movements are more brisk ; and although air may not be taken in more frequently or in such great quantity as in other cases, yet it enters more suddenly, and meets with greater resistance in its passage, so that it must cause more sound. As this loud respiration is commonly met with in children, Laennec called it puerile respiration. So also, by rendering the respiration quicker and more energetic, it may be made to sound loud in those cases in which it is naturally faint ; as by the quick short breathing just mentioned ; or better still, by desiring the person to hold his breath for a while ; the quick strong inspiration which follows is noisy enough. Disease sometimes brings about this same change : thus, if a considerable portion of the lungs be obstructed, the force of the act of breathing will be concentrated on the remaining portions, and the air will be carried in and out of them with unusual energy and noise. Hence Andral terms this partially increased respiration, *supplementary*. So also under some circumstances, without any obstruction, the need of breath may be increased ; as it happens during moderate exercise; in some degree during digestion and on exposure to cold : here the whole respiration is more energetic, and its sound louder. Further, as the act of breathing depends on a particular impression of the nervous system, so it may be supposed, when this system is preternaturally sensitive, the ordinary impression produces an increased effect ; here, again, the respiration becomes more energetic and noisy. This appears to be the chief cause of the increased breath-sound in hysteria and other diseases where the nervous sensibility is exalted. Lastly, it is possible, by an external restraint of some parts of the chest, to render the sound of respiration louder in other parts. Thus, on enclosing the abdomen and lower part of the chest in a tight belt, (and the experiment is already prepared in the persons of tight-laced females,) or when the abdomen is distended by pregnancy or dropsy, the breath-sound is unusually loud in the upper parts; and it may be seen by the heaving of these parts how their motions are augmented. Acute pain, or tenderness of some of the parts moved in respiration, would have somewhat of the same effect as a ligature, for it would cause an instinctive restraint of these parts, which would throw on others supplementary labour.

It appears, then, that there may be much variety in the sound of respiration without disease of the lungs; and, except in the cases last mentioned, it is where there is a comparative discrepancy in the several parts of the lungs, rather than any absolute difference, that disease of these organs is indicated. Thus, if we find the respiration loud on one side and obscure on the other, or clear in the lower part of the chest, and indistinct in the upper, we may well suspect some obstruction to exist in those parts where the sound is obscure; and the nature of that obstruction is then to be tested by percussion and other means.

There is another kind of variation in the breath-sound, which was first described by myself,—that which affects its *duration*. In this, as in other varieties, there are absolute differences in different individuals, and in the same individual under different circumstances; but I shall only notice the comparative discrepancies in the same subject and at the same time, which alone constitute signs of disease. We may hear the sound of inspiration on one side distinct and prolonged during the whole inspiratory act, whilst on the other side it is loud enough at first, but is abruptly arrested before the act is complete, and it is stopped with a sort of hitch. Hepatisation, or compression of the lower portions of the lungs, will do this; so will a moveable plug of tough mucus in the bronchial tubes. In other cases, again, we find the circumstances reversed: there is in a part of the lung no sound during the first part of inspiration, but towards its end, when the chest is most expanded, there is a short wheeze. This happens where the bronchial tubes are so far obstructed that air will not pass through them until they are distended by a full inspiration, as in bronchitis. It happens also in extensive pleuritic effusions, which distend the parietes of the chest beyond the medium state of respiration. It is only the acmé of inspiration that can then introduce air into the compressed lung, and it is at this period alone that the respiratory sound is heard.

There are other morbid varieties of breath-sound, such as the *cavernous*, caused by the passage of air in and out of an unnatural hollow or cavity in the lung; and where this cavity is very large, the sound becomes *amphoric*, like that of blowing into a phial. These and other varieties will be noticed when we treat of the diseases which produce them, and are included in the tabular view of the sounds given further on.

Modified breath-sounds—rhonchi.

We have hitherto considered the sounds produced by the passage of the air to and fro in the lungs, and we found that the varieties of these sounds depend on the size of the tubes, and on the force with which the air strikes against their sides and angles, and that they may be shortened or stopped by various kinds of obstruction.

We have now to describe a class of novel sounds, which arise from *partial obstructions* to the passage of the air,—obstructions which permit the air to pass, but not without such a resistance as causes an increased and modified sound. Thus if a bronchial tube be narrowed by the swelling of its membrane, or by mucus secreted by it, the air will pass through the narrowed portion with increased velocity and increased resistance; and hence the sound is changed from a simple breathing or blowing to a louder wheezing, bubbling, whistling, or snoring, according to the nature of the obstruction. These new sounds Laennec called *râles*, or *rattles.* If we must use a separate word, I should prefer the Latin term *rhonchus*, (which is from the Greek ῥογχος,) as more expressive, and it has been adopted by many English writers. If there were one, it would be desirable to use an English word, for nothing injures the purity of a language more than the introduction of foreign words.*

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* Every one who has been much engaged in describing the phenomena of auscultation, especially for clinical purposes, must have felt the inconvenience of the periphrastic and cumbersome language which he is obliged to use. Meaning is often obscured, and conciseness is unattainable, when we encumber every sentence with such complex nouns as "sound on percussion," "sound of vesicular

The rhonchi may be produced in inspiration, expiration, or in both. They may be divided into the *dry* and the *humid*, according as the impediment that produces them is solid or liquid. Of the *dry* rhonchi, there is the *sibilant*, or whistling rhonchus, which is sufficiently described by its name, and may generally be imitated by whistling between the teeth. It is produced by the passage of air through a small and somewhat circular aperture, and this aperture may be formed by a slight obstruction of a small tube, or by a greater obstruction in tubes of larger size. It generally occurs in tubes narrowed by swelling of their mucous and submucous coats, such as occurs in the early stage of acute bronchitis; but it is heard also in asthma, where the tubes are congested and constricted by the spasmodic contraction of their circular fibres; and it may happen also when viscid mucus clings to and diminishes the calibre of the tubes.

The *sonorous* rhonchus is a snoring, humming, or droning sound, and may vary in loudness or key from an acute note, like the hum of a gnat, down to the grave tone of a violoncello or bassoon. It must be produced by an obstruction leaving a flattened aperture, the lips of which, or the moisture on them, yield to the passing air with a vibrating resistance. Partial swelling of the sides of a tube, particularly at its bifurcation, a pellet of tough mucus in it, or external pressure on it, may cause such a flattened opening within the tube; and the sound in question, therefore, occurs in various forms of bronchitis, and often accompanies tumours which press on the bronchial tubes. When caused by tough phlegm, coughing generally changes or removes it: when from the other causes, it is generally more permanent. When quite permanent, it usually depends on the pressure of a

respiration," or "vesicular respiratory murmur," &c. The French terms are not more concise, and their seeming to us more expressive is only because we are less familiar with the common meaning of the words. Again, if we attempt to construct words from the only legitimate classical source, the Greek, we find ourselves hampered by a want of scientific precision in the meaning of certain words: instance the want of distinction between *sound* and *voice*. I really do not see why we should not follow the example of the Germans, and compound terms from the simplest and shortest words of our own language. Thus for sound on percussion I would say *stroke-sound;* for sound of respiration, *breath-sound;* for resonance of the voice, *voice-sound*. Although these terms may be less euphonous, they are more expressive and concise than those in common use; and being simple, they readily admit the addition to them of epithets descriptive of their character. I do not propose to supersede altogether the word rhonchus, because, like the French râle, it distinguishes a class of sounds produced by increased resistance to the air moving through the lungs; yet as they are all sounds of respiration, it might be easy to express them by proper epithets to the word breath-sound, such as whistling, humming, bubbling, crackling, &c.

tumour, or some deposit outside the tube. The key or note depends chiefly on the size of the aperture left: when this is small, the note is high; when large, it is more of a bass. From this it may be inferred that the latter can have its seat only in the large tubes; but as a more considerable obstruction may flatten their calibre to the smallest size, these may also be the seat of the acute notes. Almost every variety of this rhonchus may be imitated, by blowing between the lips moistened with saliva, and almost closed.

There is another rhonchus, which may be called the *dry mucous*, because it is produced by a pellet of tough mucus obstructing the tube, and yielding to the air only in successive jerks, which cause a ticking sound like that of a click-wheel. When the air is driven very fast, these click-sounds pass into a continuous note, and constitute the sonorous rhonchus. Sometimes, again, particularly in inspiration, the click-sound suddenly stops, the tough mucus being forced into a smaller tube, which it completely closes, and may not be dislodged again but by dint of forcible coughing.

Now as any of these rhonchi may be produced in only one tube, and yet be very loud, it is not to be supposed that they are important in proportion to the noise they make. It is rather when they are permanent, or when several of them are heard at once in different parts of the lungs, and impair or supersede the natural breath-sound, that they bespeak disorder that may be serious, either from its continuance or from its extent.

The *humid* rhonchi depend on the passage of air in bubbles through a liquid in the lungs, and their varieties are produced by differences in the size of the tubes, and in the nature and quantity of the liquid, which cause differences in the bubbling sound. A bubble is a portion of air contained and slightly compressed by a thin film of liquid, which preserves its integrity by its molecular or aggregative attraction: when this attraction is overcome by the gravitation of the liquid, the motion of the air, or any other disturbing cause, the bubble bursts; as it bursts, the air from it, slightly expanding, gives to the adjacent air an impulse which, if forcible enough, produces sound. In the bubbling passage of air through a liquid, the air is the moving body, the liquid gives the resistance; and in proportion as these are strongly and suddenly opposed to each other, the louder will be the sound produced. If the air pass with force, it makes most noise in a liquid of some tenacity, which offers it most resistance; but if it move slowly, such tenacity may retard the breaking of the bubbles, and therefore diminish the sound. Again, air passing through a liquid in large tubes gives most sound when the liquid is thin, because the bubbles form and burst quickly; but, in passing through very small tubes, air causes more sound with a rather viscid liquid, which, adhering to the tubes, is not carried before the air so readily as one of a thinner nature. This rule is applicable to bubbling sounds or rhonchi heard in the chest.

The *mucous* rhonchus may be heard in large and smaller bronchi down to the size of a crow's quill, and in these tubes its gurgling or crackling presents different degrees of coarseness. It is an irregular and varying sound, composed of unequal bubbles, and often interspersed with some whistling, chirping, or hissing notes. Its most common cause is acute bronchitis, which, after its onset, is attended with a secretion of liquid mucus into the bronchial tubes, and the passing of the sibilant and sonorous rhonchi of the first or dry stage into the bubbling of the second or secreting stage, is often marked by a curious combination of chirping and cooing notes, like those of birds in a bush. When the bronchial tubes become enlarged by disease, or when morbid cavities are formed by the destruction of portions of the lung, the bubbling of air through liquid in these is of the coarsest kind. It is quite gurgling; and if the liquid be scanty, has a hollow character, and is called *cavernous* rhonchus.

When there is a little liquid in the smaller bronchi, the bubbling or crackling is more regular, although the sound is weak, and is sometimes only a roughness added to the ordinary respiratory murmur. This is the *submucous* rhonchus. It may result from slight degrees of bronchitis, and owes its importance only to its being permanently present, when such slight inflammation is constantly kept up by the irritation of adjacent tubercles in an incipient state.

When there is more liquid, not viscid, in the smallest tubes and terminal cells, the rhonchus has a still more crepitating character, and resembles that heard on applying the ear near the surface of a liquid slightly effervescing, such as champagne, or bottled cider. This is the *subcrepitant* rhonchus, which is heard in œdema of the lungs, humid bronchitis, and other affections, in which liquid and air occupy the extreme tubes, and are forced through each other in the motions of breathing.

But the most perfect and equal crackling is that of peripneumony: it is therefore called the *crepitant*, crackling rhonchus. It exactly resembles the sound produced by rubbing slowly and firmly between the finger and thumb a lock of one's hair near the ear. I believe that this sound depends on the forcible passage of air through a little viscid mucus on the finest tubes, narrowed by congestion and deposit around them; but we shall have occasion to investigate this subject under the head of PNEUMONIA.

Of all these different rhonchi we may repeat what we said of the morbid sounds of respiration—that they may occupy the whole of the respiratory movements, or be confined to part of them. Thus an obstruction which is sufficient at the commencement of inspiration to cause a rhonchus, may be insufficient when the tubes are dilated by the distension of a full breath; or there may be the converse, an obstruction which is total in low degrees of respiration, and stops all sound, in forced or extensive efforts, as in coughing, occasions a rhonchus. This suggests to us the propriety of using these different degrees of respiration to test the nature and extent of bronchial obstructions. It may also be inferred from what has been said, that the different stages and degrees of force in respiration may change the note of the different rhonchi, and thus produce such a variety as that which we hear in the chests of some catarrhal and asthmatic patients. Laennec used to call this combination of piping sounds, rhonchus *canorus*. It may be readily conceived, too, that these several rhonchi may be variously combined, or exist at the same time in different parts of the lungs, and give rise to numerous combinations which it is useless to dwell on. I have stated that the loudness of a sonorous or sibilant rhonchus is no proof of the severity of the disease; nor is the fact of its being audible over the whole chest, unless the natural breath-sound be at the same time absent or very feeble in parts. But the presence of the bubbling rhonchi does imply mischief proportioned to its extent; and if they are heard over a large space, and accompany the whole act of respiration, diminishing or superseding the natural breath-sound, they denote disease of a very serious character, because, as our hearing informs us, there is an obstructing liquid in the tubes where there ought to be only air, and the function of respiration must be injured in proportion.

Auscultation of the voice.

We have now to examine another class of sounds,—those of the voice, as transmitted through the chest. We have found that the sounds of respiration, which are chiefly produced by air passing in the lungs, are transmitted to the ear on the surface of the chest. In like manner, the sounds of the voice, which are strongly communicated to the same air, are transmitted, modified by the size of the tubes, and the nature of the substance through which they pass; and thus these sounds also become signs of the condition of the organs that transmit them.

On applying the ear to the throat or upper part of the sternum of an individual whilst he is speaking, the voice is heard so loud, that it seems as if he were speaking into the ear, only the articulation is not so distinct. The reason of this is obvious: the sound of the voice, although originating in the vibration of the glottis, is propagated to the air above and below it; in that below, being pent up, it is not heard without bringing the ear into contact with the parts where the tubes run, and it there resounds with all its force. This is called *tracheophony*, or the natural tracheal voice.

But when the trachea divides and subdivides, there is not only a division of the sound into smaller tubes, and a consequent diffusion of it and reduction of its strength; but at this division the tubes plunge into the spongy tissue of the lung, which, as we have before found, is a bad conductor, and tends to stop the sound. Hence over the chief bronchial ramifications, as on each side of the upper part of the sternum, at and between the scapulæ, and sometimes in the axillæ, the voice is still heard, but more diffused and distant than at the throat and sternum, and the articulation is still less distinct. This is natural *bronchophony*, or *bronchial voice-sound.*

In other parts of the chest, as the voice gets into the finer tubes with their more flaccid coats and minute cells, its vibrations are either choked and destroyed, or in some parts they may be transmitted across the tissue to the parietes in merely an obscure diffused fremitus. This may be called the *pectoral* or *vesicular voice-sound.* Its vibrations may also be felt by the hand applied to the chest. Before describing the modifications of these sounds by disease, I must notice some natural varieties and their physical causes.

Natural bronchophony, or vocal resonance in the bronchial tubes, is most distinct in thin persons with a high or treble voice, as in females and children. Shrill or treble notes penetrate further into the small tubes, because their vibrations are less extensive, and need less room than those of deeper tone. This may be understood on observing the different vibrations of the cords of a musical instrument. The motions of the treble cords are short and quick, so as to be scarcely visible, whilst those of the bass are long and quite distinct. So in a person with a bass voice, the sound will hardly pass into the subdivisions of the tubes, and there will be little or no bronchophony; but if the voice be strong, it will not be entirely lost, for it will pass across the whole spongy tissue, and throw it all, more or less, into a diffused vibration, which may be heard and felt in many parts of the chest in the character of pectoral voice-sound. We find, then, that treble tones give more of bronchophony, and bass tones more of the pectoral voice-sound. The same occurs with the morbid sounds; and if we can get our patients sometimes to change their tone of voice, we may thereby more effectually test the condition of their pectoral organs.

Now, as with corresponding varieties of respiratory sound, so with these sounds of the voice, they become signs of disease when they are heard out of their proper places. To know what these proper places generally are, it is necessary to study the anatomical disposition of the tubes and tissue of the lungs in the different regions. But there is another standard more applicable to individuals—this is, comparison between the two sides of the chest. As there is an approach to symmetry in the structure of the bodies, so there is, in health, a general correspondence between their sounds; and as disease scarcely ever affects both sides at the same time and in the same degree, it will make the phenomena of one side to differ from those of the other. For example, if under one clavicle the voice resound loudly, whilst it is scarcely heard under the other, it is certain that there is some physical difference between the two sides that does not exist naturally; or if below the third rib in front there be heard the tubular or bronchial voice, which is generally confined to the immediate neighbourhood of the large bronchi, it may be inferred that there is an altered condition of the parts. Let us inquire what alterations will change the natural disposition of the sounds.

An increase in the density of the pulmonary tissue by a solid or liquid effusion, or even extensive sanguineous congestion in it, will improve its condensing power, enable it to transmit from the bronchial tubes within, the vocal sounds which they receive from the trachea. This is morbid bronchophony, or *bronchial voice-sound*, and it is often, but not always, accompanied with bronchial respiration. If, then, the voice be heard resounding in a part of the chest where it is not usually heard, it may be suspected that the lung is in some way increased in density; but this is not certain until it be tested by further means, for there is another change which may also increase the vocal resonance of a part. If, instead of the sound being better conducted from within, it is increased in strength and extent by an enlargement of the bronchial tubes, it may then be heard in situations where it does not naturally reach the walls of the chest. In both cases it may more or less resemble the natural bronchophony heard near the top of the sternum and between the scapulæ; but it often presents considerable modifications. Thus, when transmitted from the middle-sized bronchi, it comes rather as *diminutived bits of voice* than as articulate words. This may be called slender bronchophony. In this variety, for reasons before mentioned, low tones are not transmitted; so that if the patient varies his cadence, some words are heard and others not. When arising from dilated air-tubes, or when transmitted from the larger tubes, the bronchophony is more noisy and continued, varying less with the tone of the voice. If the air-cells over the resonant tubes be still open, the sound will be diminished when they are dilated by a full inspiration, because they then tend to intercept it more, and the variations caused by these motions, together with occasional whiffs of bronchial breath-sound, may produce the varieties of bronchophony called by Laennec *puffing* and *veiled.* The loudest bronchophony is caused where the middle and upper lobes of the lung are pressed against some part of the walls of the chest by a liquid effusion in the pleura, which cannot displace the lung from that part, because it is bound to it by old adhesions. In such cases, the stroke-sound is commonly also tubular.

But what modifies the transmitted voice in the most remarkable manner, is a thin layer of liquid between the lung and the walls of the chest. The liquid is thrown, by the vocal resonance of the lung, into a state of irregular vibration, which causes it to transmit the voice in a broken, tremulous manner, so that it sounds to the ear outside like the bleating of a goat. Hence Laennec called it *ægophony* (αιγος φωνη). In conformity with the other terms, it may be named *bleating voice-sound.* It may be produced simply by liquid in the pleural sac, without disease of the lung; for the compression of the pulmonary tissue caused by the liquid is enough to enable the lung to transmit the voice from the bronchial tubes within it. When the liquid is increased, the sound becomes more distant and silvery, like a slender echo of the real voice. When the lung is consolidated also by disease, the vocal resonance is stronger, and there is a loud bronchophony mixed with the bleating voice, constituting a kind of double or buzzing voice, which Laennec compared to that of Punch, as performed in the streets.

There is yet another kind, which may be called the perfection of pectoral voice-sound. When a cavity is formed in the lung by the emptying of a vomica or abscess through the air-tubes, the voice passes from these tubes into it; and if the communication be free, the voice may, by the ear applied outside, be heard in the cavity as distinct as it is in the trachea. This is *cavernous voice-sound,* or *pectoriloquy*—not only voice, but *speaking* in the chest. When the cavity is near the surface, of a moderate size, and opens freely into a large air-tube, the phenomenon is most perfect, and then sounds exactly as if a patient spoke into the ear; this is limited to the spot where the cavity lies, which is thus, as it were, a little island of voice, and is a sure sign of a cavity. The sound of bronchophony is often louder, but then it is more diffused, and there is less distinctness in the words. We shall enter further into these distinctions when we treat of the lesions of which they are signs.

When the cavity is large, and the opening into it small, the voice may not fully enter it; but there may be a tinkling, or hollow reverberation in it, like that in a phial. This is an echo, modified by repeated reflection, and constituting a separate note. It is called *amphoric resonance*, or *metallic tinkling*, according to the character of the sound. It may be produced in the cavity left by a large vomica or abscess, or by several of these running together, but its more common seat is the sac of the pleura, into which the air has entered through a fistulous opening from the lung. Now this being the resonant or echoing cavity, it is plain, that not the voice only, but the breathing and cough also, especially if they be accompanied by a bubbling through the fistula, will have more or less of this tinkling or hollow sound.

Besides these various positive phenomena of the voice, the *absence* of the pectoral voice-sound is sometimes a valuable sign. I have stated that this vocal vibration can be felt as well as heard. On applying the hands, one on each side of a healthy chest, the vibrations may be felt nearly alike on both sides. Liquid in the pleura will generally, more or less, destroy this fremitus; and the difference which it produces between the two sides is often a very valuable sign of the presence of liquid. Consolidation of the lung, again, will increase the vibrations, or make them even stronger over the bronchial tubes. In cases where one side is quite dull on percussion, we may often thus easily distinguish whether the dulness is caused by consolidated lung or liquid in the pleura, which is a point of great importance.

Pleural Friction-Sound.

Besides the sounds produced by air and the voice, there is sometimes one produced by the motion of the lung against the ribs. The lungs, although they nearly follow the motions of the chest, do not move quite with it, especially in the lower parts, where the descent of the diaphragm draws the lungs downwards whilst the ribs are rising. But in the natural condition, the surfaces of the pulmonary and costal pleura are so smooth, and so well lubricated with serum, that although there is motion, there is not resistance enough to that motion to cause sound. But if these surfaces become uneven by the deposit of rough or adhesive matter on them, or by an irregular distension of the tissue by solids or air under them, there may then be a rubbing sound with the motions of respiration. This occurs in pleurisy, and, according to Laennec, in emphysema of the lung. This rubbing sound is often the more evident in these cases, because the same disease, by preventing the proper expansion of the lung, causes less harmony than usual between its motions and those of the chest.

The foregoing description of the acoustic phenomena of the chest, connected with respiration, may perhaps be considered rather minute; and it is hardly expected that the student will be able easily to master all the details; but if sufficient attention be paid to the principles that have been explained, the various phenomena which are illustrations of these principles will become familiar and intelligible when they present themselves in clinical experience. I would rather not encumber the mind of the student with too many details; but I do hold it to be highly expedient that he should be well acquainted with the laws under which they occur; for from these and a good knowledge of pathology, he may understand and deduce, not only the phenomena hitherto observed, but new forms and combinations of signs, which observation and practice may bring before him. The subjoined tabular view of the chief phenomena of auscultation of the organs of respiration may give additional assistance.

SOUNDS PRODUCED BY THE PASSAGE OF AIR IN RESPIRATION.

Sound of Respiration, or Breath Sound.—

Natural; produced by collision of the air against the sides and angles of the air tubes.

Tracheal; heard in the neck and at the top of the sternum.

Bronchial; near the upper parts of the sternum, between the scapulæ, &c.

Vesicular; in most other parts of the chest.

Morbid, modified in production or transmission.

Bronchial, or whiffing; transmitted from the bronchi by condensed tissue of the lung.

Cavernous
Amphoric } produced in morbid cavities communicating with the bronchi.

Rhonchi, produced by increased resistance to the air moving through the lungs.

Dry;	Sibilant Sonorous Dry mucous	produced by viscid mucus in the bronchi, or by swelling of the membranes, or by pressure upon them.	
Moist;	Mucous . .	produced by a bubbling passage of air through	liquid in the bronchi.
	Submucous		a liquid in the finer bronchi.
	Subcrepitant		liquid in the smallest bronchi.
	Crepitant . . . , ...		viscid liquid in compressed smallest bronchi.
	Cavernous		liquid in a morbid cavity.

SOUNDS OF THE VOICE TRANSMITTED THROUGH THE CHEST.

Natural Sounds, *heard in a healthy chest.*

Tracheophony, in the neck and at the top of the sternum.

Bronchophony, near top of the sternum, between the scapulæ, in the axillæ, &c.

Pectoral voice-sound, in many parts of the chest.

Morbid Sounds, transmitted or produced by a diseased chest.

Bronchophony, transmitted by condensed pulmonary tissue.

Ægophony, the same vibrating through a thin layer of liquid.

Pectoriloquy, resounding in a cavity in the lung.

Tinkling, a changed echo of the voice or cough in a large cavity.

SOUNDS PRODUCED BY THE MOTIONS OF THE LUNGS.

Sounds of friction, when the pleuræ are dry, or rough from deposits.

Emphysematous crackling, by the irregular passage of air between the lobules.

Methods of Auscultation.

I have now to describe shortly the methods of auscultation, or the means which we use to obtain a cognizance of those acoustic phenomena which we have found to be signs of the condition of the organs within the chest. We have already described the method of percussion; and we have now to study the best mode of listening to the signs of the motions of the chest.

All these signs can be heard by the direct application of the ear to the chest; and this *immediate* method of auscultation is so easy and simple, that it commends itself strongly to us, and in many cases is used with advantage. The sounds proceeding from the walls of the chest are communicated to the ear, and especially to the air contained in the external meatus, and are thus propagated in the most direct and unmodified manner to the organ of hearing. *Immediate* auscultation is exclusively practised by some, both at home and abroad; and as it is much more easily learned than the *mediate* method, it will probably always have its advocates among those who prefer ease to exactness. And if we can hear the signs so well by the unassisted ear, it may be asked, what is the use of the *stethoscope?* I shall first mention some positive objections to immediate auscultation; and, on examining the principles of the stethoscope, we shall find that it has, in many cases, considerable positive advantages. To apply one's ear, and therefore the nose, face, and so forth, to the chest of a patient who is dirty, blistered, or wet with perspiration, would be disgusting. To apply it to the chest of a patient labouring under an infectious disorder would be unsafe. To apply it to the person of a young female would scarcely be delicate. Moreover, it is difficult to apply the ear well to some parts of the chest, such as the armpit, and below the clavicles or between the scapulæ, in thin persons. Besides this, disturbing noises sometimes arise, from the contact of one's hair or clothes with the patient's chest; and unless the observer's neck be pretty long and flexible, this *easy* method will be found, after all, more fatiguing than the mediate method. Still, in a great many instances, it may be used with advantage, especially in examining the regions of the back, and in children, where the stethoscope might cause alarm, and could not be so steadily or quickly applied.

We want an instrument, then, to transfer the sounds from the chest to our ear. This instrument must be a good conductor of sound; and as the power of bodies to conduct sound depends on the strength and uniformity of their elasticity, and their capacity to vibrate like the body that produces the sound, we must have an elastic material, of density resembling that of the sources of sound within the chest, and of the walls of the chest through which they are transmitted. But the sources of the pectoral sounds vary. Some, as the voice and respiration, or at least the hollower sounds of respiration, are produced in air, whilst in others, such as the sonorous rhonchus, the rubbing sound, and the sounds of the heart, the solids are chiefly concerned. We shall, therefore, need a varied capacity in our instrument, to receive these sounds. It has been objected to this view, that as all these sounds have to pass through the solid walls of the chest, their differences must here cease; and their character being identified by being made vibrations of solids, the instrument need only be adapted to these solids. But the vibrations do not always change their character with every change of medium; and the elastic, yielding walls of the chest do not considerably modify the vibrations which they receive. As we found by percussion that they derive the character of their vibrations from the nature of the material which lies under them, so when this material originates a sound, the walls readily vibrate with it, without materially changing its nature or the manner in which it may be communicated to other bodies. It is thus that one parchment head of a drum transmits its vibrations through the air, rather than through the solids, to the other head, which in its turn, although a solid, freely transmits its motions to the air. Solids, when thin, instead of vibrating only according to their own molecular elasticity, are readily moved or carried by the vibrations of the media on either side of them, and their vibrations partake of the character of those of such media. This is the reason why we hear voices easily through the thin partition of a room; the sound passes from the air to the partition, and from the partition to the air, and would not pass so readily if for one portion of the air were substituted a conductor of the same density as the partition itself. This point might be further illustrated; but enough has been said to show why our instruments for conducting sounds from the chest to the ear should be constructed with regard rather to the origin of those sounds, than to the density of those solid walls through which they pass.

To transmit the sounds originating in the solids within the chest, we must have a uniform solid, and the lighter it is the better, provided it be thoroughly rigid. Now nothing answers to this description so well as wood; and in the light kinds of wood, with a stiff, longitudinal fibre, such as pine-wood, deal, cedar, and the like, we find these qualities in perfection. Through a cylinder of such wood, about eight inches long, and an inch and a half in diameter, adapted to the ear at one end, most of the pectoral sounds may be heard, but those best which originate in solids, such as the sounds of the heart, of friction, and sonorous rhonchi. The sounds of respiration and of the voice are also heard through it, but not nearly so distinctly as with the naked ear.

We need, therefore, an aerial conductor for these sounds, because they originate in air, and can best be transferred through air. By perforating the cylinder with a bore, a quarter of an inch in diameter, it becomes a tube, through the columns of air in which the respiration and voice may be heard with increased distinctness. But as this column of air is in contact with only a small spot of the chest, it can transmit only the sounds produced under or very near that spot, and the instrument thus prepared is better adapted to explore small parts of the chest than the ear can be. But we want the instrument also to transfer the sounds of larger spaces. The sounds of so limited a space are often too weak to be heard alone; and, besides, it would be very tedious to go over the whole chest, *dotting* in this way a quarter of an inch at a time. Now, if the column of air be enlarged at the base, where it is in contact with the chest, by hollowing out the wooden cylinder into a funnel shape, it will conduct the sounds produced on this greater extent of surface, which are reflected by the funnel into this central bore, and conveyed concentrated to the ear. This also gives the instrument the power of concentrating or magnifying the sounds; they are thus heard as strong at the distance of several inches, or even a foot or two from the chest, as they are to the ear in close contact with it; nay, in some cases they are even stronger. The best shape for the excavated end is that of a long funnel or cone, with its apex terminating in the central bore; for this directs the sound at once to the ear, without repeated reflections, which may modify it. As we still sometimes want to explore small spots of the chest, by means of a perforated plug the excavated end can be filled, and the instrument reconverted into a simply perforated cylinder. To make the instrument more portable, the upper part of the cylinder may be reduced to a stem half an inch or less in diameter, leaving only at the top a sufficient width for the ear; or this top may be made of a harder wood. Wood is so excellent a conductor of sound, that when once the vibrations are in it, they can be conveyed by a very small body of fibres.

Thus the stethoscope, although a simple instrument, performs several offices in relation to sound, the chief of which may be enumerated as follows:—

1. To conduct sound by its solid walls.
2. To conduct and concentrate sound by its closed column of air.
3. To transfer sounds from its column of air to its solid walls, or the converse, when circumstances impede their transmission by one of these ways.
4. To diminish this power of transfer, and contract the field of hearing when small spots are to be explored.*

We have now only to add a few words on the method of using the stethoscope. It is quite necessary that the instrument should be applied in close contact with the chest and the ear; the least tilting uncloses the column of air, and occasions great loss of sound outwardly, as well as a confusing entrance of extraneous noises. To prevent this tilting, it is best first to apply the stethoscope, held by its pectoral end, firm on the chest, and then to apply the ear flat on the top. If the inequalities of the ribs leave chinks between the chest and the instrument, a fold or two of linen will fill these, or the stethoscope may be used with the stopper in. For the sounds generally, it is better to use the instrument without the stopper; but when it is an object to determine whether a sound is produced in a limited space, or over some extent of surface, the circumscribing power of the stopper is wanted. Thus it is often of importance to determine whether a local resonance is produced in a small cavity, or merely transmitted by consolidated lung from several bronchial tubes, distributed over some extent of surface. The simply perforated cylinder will often do this by showing the size and shape of the limited spot in which the voice-sound, or pectoriloquy, of a cavity can be heard in its full strength, while the bronchial voice-sound is transmitted less strongly, and may be traced over some extent of surface, generally in the known direction of these tubes. The stopper is also useful in shutting out the breath-sounds, where it is an object to listen to the sound of the heart or arteries; and in many other circumstances, which will be noticed in the history of individual diseases. When we wish to listen to the voice in the chest, the patient should be desired to turn his face away from the auscultator, and answer questions, or count from twenty, so as to speak several consecutive syllables. To make the sounds of respiration distinct, it is often necessary to make the patient breathe quick or to cough, and in some cases to hold his breath for a few seconds, and then to take a deep breath.

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* Dr. George Budd (Med. Gaz., May 27, 1837) has advanced an opinion that the solid material of the stethoscope is the sole conductor of sound in the stethoscope, and that the advantage of the hollow is merely to give the solid a thinness and lightness, which make it more susceptible of vibration. This notion has been advocated by Dr. Cowan, (Med. Gaz., Oct. 28, 1837,) who relates several experiments to support it. Of these only one has any force. He states, that "If one or both ends of a stethoscope are accurately closed with cork, the respiratory murmur is only *very slightly* weakened." This experiment has been repeated by several competent observers, as well as by myself, with a different result—all have agreed that the sound was thus *considerably* impaired. The experiment may, however, be more fairly tested on the flexible ear-tube used as a stethoscope, which, according to my views, conveys sound almost entirely by its enclosed column of air; but, according to Drs. Budd and Cowan, it too conducts only through its solid walls. *The cork here will be found almost wholly to intercept the sound.* With this instrument, too, I have often heard the sounds of the heart and of the voice, when the ear-piece is only approached to the ear without contact. So, in like manner, with a common stethoscope, very loud cardial murmurs may sometimes be heard, by placing the ear close to the ear-piece without contact. In both these cases, the transmission is *wholly prevented* by a plug in the tube. All these facts show the important share which the column of air has in the conducting properties of the stethoscope. No doubt the solid conducts also, as I have always supposed; and, as Dr. Cowan suggests, it does so the more readily in proportion as its mass is lighter and its surface is increased. This is in conformity with the law of sounding-boards, which I had formerly explained and applied to the case in question.

In conducting physical examination, due care should be taken to avoid fatigue or annoyance to the patient. There are cases in which a complete physical examination will do more harm than the information which it may bring can do good: but they are few, and it must be left to the discretion of the practitioner to hold the balance between too much and too little examination. Experience soon points out that the observer must also consult his own ease in the act of auscultation, for a constrained or painful posture impedes the hearing and disturbs the attention. For this reason it is sometimes easier to hear with a flexible ear-tube than with the straight stethoscope, although the latter is by far the best instrument for general purposes.

CHAPTER II.

EXAMINATION OF THE CHEST THROUGH THE VITAL PROPERTIES OR FUNCTIONS OF ITS ORGANS—ANALYSIS OF THE GENERAL SYMPTOMS OF DISEASES OF THE CHEST.

We have been occupied in the preceding pages in considering the physical properties of the chest and its organs, and the manner in which these properties may become signs of the condition of these parts. We have now to examine them through their *vital* properties, which, combined with certain physical and chemical powers, constitute *function.* Physiology teaches us that the elementary vital properties immediately concerned in the function of respiration, are sensibility, contractility, and the power of secretion. These properties are linked together with the chemistry and mechanism of the organs of respiration, so as to constitute their healthy function. Now any excess, defect, or disorder of any of these properties, will be more or less felt throughout the links of this chain; and hence may arise, not only derangement of the function of respiration, or *dyspnœa*, but also new phenomena, proceeding from a loss of due balance of the properties, such as *cough*, *expectoration*, and *pain ;* and associated as the vital properties are with those of other organs, there may be added disorders of these, in the form of *disturbance* of the *circulation*, and its sign the *arterial pulse*, *general fever*, *disorder of the secretions of the kidneys*, *liver, and intestines*, and of the *digestive*, *nutritive*, *and sensorial functions.* The phenomena arising from these several disordered properties are what are called the *vital* or *general symptoms* of disease, which we now have to consider in relation to the organs of respiration.

Now it might be inferred, and it will be more apparent as we proceed, that these general symptoms, dependent as they are on such a linking together of many properties, the laws of which are but imperfectly understood, must be far less simple and intelligible than the physical signs; and the variable measure of the vital properties also renders general symptoms far more uncertain than these signs, in their degree, and even in their presence. We cannot with any certainty, as with the physical signs, from a knowledge of the phenomena, and the laws which regulate those phenomena, deduce the condition of the parts which produce them, nor, from knowing the condition of parts and physical laws, deduce what phenomena the parts ought to develope. For example, the solids of the body have sensibility, which varies not only in different parts, but in the same parts at different times, and this for reasons which we cannot discover; therefore we cannot calculate on it. The contractility of moving parts also varies in a similar manner: and we can by no means gain, from the character of their motions, a criterion of their true condition. Instead, therefore, of pursuing the synthetic as well as the analytic method, which we have done with regard to the *physical* examination of the chest, we shall shortly analyse the chief *general symptoms* of diseases of the chest, and by that examination endeavour to determine their nature and varieties, and their value in teaching us to discover, to measure, and to treat these diseases.

Dyspnœa, difficult or disordered breathing, is the most important general symptom of disease of the chest, inasmuch as it implies more or less interruption to the due performance of some part of the great function of the chest—respiration. Dyspnœa may be caused by circumstances affecting any one or more of the several elements concerned in the function of respiration: viz. the blood in the lungs, the air, the machinery of respiration by which these are brought together, and the nervous system through which the impression which prompts the respiratory act is conveyed from the lungs to the medulla oblongata, and thence to the muscles which move the machinery: in fact, all the causes which in excess produce asphyxia, in slighter degrees occasion dyspnœa. The opposite table exemplifies these causes of dyspnœa; but let us now attend to the character of the symptom itself.

When any thing interferes with the sufficient action of the air on the blood, the impression which excites the acts of breathing not being relieved, causes a quicker and fuller repetition of this act; and if the interference still remain, the breathing will continue to be more or less hurried and forced, until the sensation or impression is reduced to the ordinary standard of almost unconsciousness. Now an individual in whom the breathing is hurried may not be sensible that it is accelerated; whilst, in another who feels the oppression, there may be little appearance of shortness of breath. Again, the feeling of dyspnœa must greatly depend on the

PROXIMATE CAUSES OF DYSPNŒA, OR DIFFICULT BREATHING.

1. By impeding the access of pure air to the lungs.			
a. *Mechanical.*			
Rigidity of parts of the respiratory machine	*e. g.*	Ossification of cartilages; induration of the pleura; rickety distortions.	
Pressure on ditto	*e. g.*	Tumors or dropsies of the abdomen.	
Obstructions of the air-tubes	*e. g.*	Effusions in, swellings of, tumors pressing on, the air-tubes; Spasm of the glottis; spasm of the bronchi.	
Compression of the lungs	*e. g.*	Effusions or tumors in pleural sac	Pleurisy, Hydrothorax, Pneumothorax, Aneurism, &c.
Alterations in the tissue of the lungs	*e. g.*	Engorgement.	
		Effusions	Œdema, Hepatisation, Tubercle, &c.
		Altered structure	Emphysema, Dilated bronchi, Vomicæ, &c.
b. *Chemical.*			
Deficiency of oxygen in the air	*e. g.*	Mephitic gases; rarefied air.	
c. *Vital.*			
Pain of parts moved in respiration	*e. g.*	Pleurodyne; pleuritis; peritonitis, &c.	
Paralysis of muscles of ditto	*e. g.*	Injuries of the spinal marrow in the neck, &c. Paralysis of the bronchi (?)	
Weakness of ditto	*e. g.*	Excessive prostration, from ataxic fevers, &c.	
Spasm of ditto	*e. g.*	Tetanus; spasmodic asthma, &c.	
2. By the state of the blood.			
a. *Mechanical.*			
Obstruction to the passage of the blood	*e. g.*	Diseases of the heart and great vessels; tumors pressing on them.	
b. *Chemical.*			
An excessively venous state	*e. g.*	Violent exertion; idiopathic dyspnœa (?)	
Deficiency of red particles	*e. g.*	Anæmia; chlorosis.	
3. By the nervous relations of respiration.			
Excessive sensibility of the par vagum	*e. g.*	Hysteric dyspnœa; cerebral fevers; neuralgia (?)	
Defective ditto	*e. g.*	Coma; narcotism, &c. (breathing slow.)	

condition of the sensorium; for whilst some patients are conscious of the slightest infringement on their respiration, others, particularly in congestive fevers, are brought to the verge of asphyxia without complaining of any oppression. So, too, we are sometimes astonished to find, on opening the bodies of the dead, a whole lung diseased, or one side of the chest full of serum, where the patient had not complained at all of dyspnœa: while, in other cases, a much smaller lesion of the organs has been attended with the most distressing orthopnœa. It is, however, rather to the sensation of breathlessness than to merely accelerated breathing, that the word dyspnœa is generally attached: for, translating it as difficult breathing, this expression can be hardly applied when the difficulty is overcome by accelerated movements, of which the patient may not be conscious. But we shall here advert to frequency of breathing, as well as the feeling of dyspnœa.

The number of respirations in a healthy adult male at rest generally ranges about twenty in a minute. It is more in children and in females, and it becomes increased in all cases, not merely from affections of the lungs or connected organs, but also from general weakness or depressing causes, which, diminishing the strength of the muscles of respiration, oblige them to make up by the frequency of their contractions what is wanting in their energy. Probably there are some nervous conditions of the system also, in which the breathing becomes accelerated from increased *sensibility*, and from what Cullen called *mobility*, a greater readiness to move, than power to complete the motions. I have seen the breathing hurried in some cases of hysteria, without the patient being conscious of it, and without either real weakness or pectoral disease to account for it. These cases should be known, that they may be separated from those of true dyspnœa. In many other cases, especially those, I believe, where the nervous system is affected, the breathing is not accelerated, but suspirious, a sigh or deep breath being taken from time to time; yet the patient is often not conscious of any oppression or unusual effort. This may be called irregular breathing, and there are several other varieties, which it is not necessary to consider in detail. The rhythm in breathing probably depends entirely on the chain of influences which we before described as concerned in the act, and not on any peculiar periodicity, such as that which seems to reside in the heart; and therefore irregular breathing must depend on a change in one or more of the links in that chain.

The feeling of dyspnœa is one of a very peculiar and distressing character. Even when slight in degree, its permanent oppressive influence is very wearing; and when severe, it causes the most horrible suffering, with such a feeling of impending death, that the most courageous are often unmanned by it. The constrained postures of the patient, the anxious or even desperate expression of his countenance, the painful straining of all the muscles that can in any way, however distantly, assist in the respiratory movements, bespeak the intensity of the feeling, which I have heard several patients say is far worse than the most acute pain. It is worthy of remark, however, that this feeling is experienced in its severest degrees only by those in whom the dyspnœa comes on rather suddenly, especially when the sensibility is entire, and the lungs are not diseased, as in obstructions in the trachea or large bronchi, spasm or swelling of the glottis, and spasmodic asthma. In these cases the sensibility is not gradually blunted by the circulation of imperfectly oxygenated blood; nor has the activity of the functions, which require arterial blood, been lowered by previous depressing causes. Opium, belladonna, camphor, and other narcotics, will sometimes relieve the symptom of dyspnœa, not only by deadening the sensibility, but also by diminishing the activity of those functions and secretions which require oxygenated blood, and therefore a free supply of air. If we could temporarily produce a state approaching to the torpor of hibernating animals, we might diminish the bad effects, as well as the painful feeling of dyspnœa; and I believe that such a state is actually induced in those who are habitually asthmatic, in whom all the functions are brought to a lower standard, and who thus suffer with impunity such an encroachment on the function of respiration as would be fatal to an individual of a common standard.

The feeling of want of breath has been used as a means of testing the condition of the respiratory organs. A person whose respiration is free and unembarrassed, can hold his breath longer than one whose lungs are diseased. Dr. Lyons has proposed to measure the condition of the lungs by the time which a patient can hold his breath after a full inspiration, and, to insure accuracy, he is desired to count numbers during this time. A healthy person with a good chest can continue counting for forty-five seconds without taking breath, whilst those with diseased lungs often cannot keep on for twenty seconds. The same objection may be made to this test that we made to the measuring of the exhaled air, proposed by Abernethy: that it is a test as much for the strength of the muscles of respiration as for the condition of the lungs. Besides, both the feeling of wanting breath, and the power of augmenting the respiratory movements, vary considerably in different healthy subjects. It is well known that divers acquire the power of remaining under water for two or three minutes (it has been said more) without taking breath. In diving animals there is a structural provision to enable them to continue some time without air. The chief venous trunks are very tortuous, and admit of dilatation, so that the venous blood can accumulate in them, instead of distending and embarrassing the right cavities of the heart and the lungs. Perhaps some change of this kind may be somewhat produced in divers, by the often repeated practice of holding the breath. Professor Faraday has described another mode by which a person may be enabled to hold his breath for a minute and a half, which is double the time usually practicable. This is by making in succession five or six full forcible inspirations, which seem to so completely change the air in the lungs, that there is left in them a stock of pure air capable of lasting during that time. The knowledge of this fact may be useful, if ever it is required to hold one's breath for a time in going into the suffocating atmosphere of a sewer, a mine, a house on fire, or the like, or in diving.

Dyspnœa is often a symptom demanding great attention in diseases of the lungs; but it must be studied in conjunction with the other general symptoms and the physical signs, for in itself it is most vague and inconclusive. This may be perceived on inspecting the tabular view of the causes of dyspnœa, which is founded on the physiology of respiration. This table deserves attention, not only in showing the varied nature and origin of the symptom, but also in contrast with the tables of the physical signs, the causes of which are much less varied, and far more appreciable. But when through the means of the physical and other general symptoms, it has been made out on what cause the dyspnœa depends, then this symptom often becomes a valuable measure of the increase or diminution of the disease, and a useful guide of practice.

Let us now attend to another symptom, which is even more common in diseases of the chest than dyspnœa is,—I mean the *cough*. The act of coughing consists in one or more abrupt and forcible expirations, accompanied by a contraction of the glottis, trachea, and upper bronchial tubes. The expirations being more complete than usual, especially when there are several of them, are followed by a deep forcible inspiration, the force of which is shown by the loud respiratory murmur, which, by the ear applied to the chest, may be heard to accompany it. The muscles chiefly concerned in the act of coughing are the abdominal muscles and intercostals, the combined attraction of which effects a strong pressure on the contents of the chest.

The common cause of cough is phlegm or some other matter irritating the air-passages, and the object or final cause of the cough is to expel or expectorate this matter. I believe we may fairly say that the proximate cause of cough is always some irritation, either direct or by sympathy, of the sentient parts of the air-tube, or of the nerves which render them sentient. Some parts of the bronchial membrane are much more sensitive than others; that which lines the glottis and larynx is excessively so, and the least irritation of it is enough to excite coughing. That of the trachea and large bronchi is less so, for foreign bodies have been known to lodge in them some time without causing any coughing, so that some have supposed that they have nothing to do with the production of this symptom; but when the sensibility of these parts is increased by inflammation or nervous excitement, anything irritating them will also excite coughing. We can easily see why the sensibility of the air-tubes should be greatest at their entrance: it is the *doorkeeper*, placed there to exclude, or, by calling other forces to its aid, to expel anything improper which may intrude. But the other parts of the tubes have also a preserving sensibility, which may bear a little, but is soon roused into activity by a continued irritation. We find the parallel of this in the alimentary canal in the natural state. The sensibility that excites the action of vomiting is peculiar to the fauces at one end of the tube; and that which induces the striving of defecation resides chiefly in the termination of the rectum at the other end; but uncommon degrees of irritation, or an exalted sensibility, will occasion the same actions to be excited by impressions on other parts that are usually insensible: hence arise the vomiting caused by an over-irritated or inflamed stomach or duodenum, and the tenesmus and purging excited by a similar state of the colon. We shall see this more fully on considering the various causes of cough.

As other irritations, cough may be excited either by an unusual irritant acting on the tubes in their natural state, or by the ordinary circumstances, which, although not usually irritating, yet become so by the exalted irritability of the tubes; or, as is the more common case, by a combination of these causes. We have an example of cough excited simply by an unusual irritant, when a portion of food or of bronchial mucus lodges on the membranes of the glottis; and an irritant may act by sympathy as well as by direct application, as when we excite coughing by introducing a probe pretty far into the ear. The cause by increased irritability is exemplified in the cough of early bronchitis and nervous asthma, which the mere inhalation of air is sufficient to excite. There are both an unusual irritant, and increased irritability in the secreting stages of bronchitis, and other affections, where an unusual quantity, and sometimes an irritating kind, of mucus is poured out on an over-sensitive membrane. This more complex cause of cough is frequently induced by the continuance of the other causes; thus, the continued application of an irritant will develope an increased sensibility, and increased sensibility and irritation will be followed by inflammatory excitement and the secretion of matter, the quantity and quality of which add to the irritation. Thus we see how the physiological causes of cough become identified with the pathology of bronchitis, or inflammation of the membrane of the air-tubes; and, in common parlance, a bronchial inflammation is called a *cough*, this being the most prominent symptom. But although this inflammatory condition is often developed by the continuance of causes which produce cough, yet it is not necessarily so, and there may be irritation or increased sensibility, or both, enough to cause cough, and which may yet be short of the degree or the conditions requisite to produce inflammation.

I have said that the irritation which causes cough may not be applied to the bronchial membrane itself, but may be exerted from a part more or less distant. Thus cough may be excited by tubercles in the parenchyma of the lungs, by inflammations or irritations of the pleura, peritoneum, stomach, liver, and so forth; and although we may conjecture that these irritations are conveyed through the nervous branches which connect these several organs and the air-tubes with one common sensitive centre, yet we cannot explain why they should be sometimes conveyed, and at others not; for although cough does frequently accompany the pathological conditions to which I have just adverted, yet irritations and inflammations often arise in the stomach, liver, peritoneum, nay, sometimes even in the pleura and pulmonary parenchyma, without any cough whatever. I have heard some one try to explain these discrepancies, by assuming that there must be bronchitis present to produce cough, and that when these several distant irritations do not excite bronchitis, they are unaccompanied by cough; but this view increases the difficulty instead of diminishing it, for it leaves unexplained the reason why this supposed bronchitis should occur in some cases and not in others; and bronchitis, although including cough, is more than cough, and needs something more to produce it. We may conjecture about local weaknesses, constitutional peculiarities, and irregular sensibilities, as causes of these differences, and this is all that we can do towards explaining them, but this is not what an explanation ought to be; this is referring phenomena, not to known general properties, and the laws which govern them, but to individual peculiarities and undefined influences, the laws of which are not known. These considerations furnish another proof of the uncertainty of general symptoms, as means of diagnosis. Still, when cough does occur, and its cause has been made out by the aid of other signs, it deserves attention, not only as a symptom, but as a morbid action of a distressing and hurtful kind, which sometimes may require remedies expressly to relieve it. This illustrates what I have before said, that general symptoms, although much less constant and instructive than physical signs, with regard to the diagnosis of organic lesions, yet, when positive, often tell us more of those general conditions of the system, which become our guides in the employment of remedies.

Under this impression, we shall examine some of the varieties of cough which present themselves in different cases, and trace the connexion between their characters and the variations in the elements that constitute them. Of course the study of a symptom in any individual case must be conjoined with a proper survey of its functional or organic cause; but as we have also (sometimes only) to treat the symptom, it is highly useful to study its varieties, and thus to render it more practically instructive.

The cough may vary according to—

1. The irritant exciting it.
2. The sensibility feeling the irritation.
3. The movements thereby excited, (*a*) the contraction of the muscles of respiration, and (*b*) the contraction of the air-tubes.
4. The condition of the bronchial membrane and its secretion.

Under these heads, we shall meet with varieties of cough with which every practitioner is familiar.

1. When other things are equal, the violence of a cough will be in proportion to the degree of irritation that excites it. For example, a healthy person, whilst eating ordrinking, incautiously suffers some food to enter the glottis; the cough thereby excited will be more severe with wine or anything peppered, than with water or any bland food. So in the early stages of catarrh, although the sensibility of the membrane is increased, yet the thin saline-tasted secretion also acts as an unusual irritant upon it, and keeps up a short, teasing, tickling cough, with continued attempts to clear the throat. When the irritation is more moderate, but irremovable, like that occasioned by incipient tubercles in the pulmonary tissue, the cough will generally be of that slight constant *hacking* kind, with little or no expectoration, that is so well known as one of the first symptoms of pulmonary consumption. The irritant here remaining the same, the circumstances which increase this cough are those that augment the sensibility of the lung and air-tubes; such as a quickened state of the circulation from exertion, heated rooms, or during the assimilation of stimulating food.

2. We have already adverted to increased sensibility as being concerned in the cough of recent bronchitis or bronchial irritation. It becomes, however, more developed when the cough has lasted several hours, and, instead of being short and tickling only, it comes on in more violent and prolonged fits, which are quite irresistible, and often accompanied by a feeling of soreness. The heightened sensibility of the air-tubes is further manifested by the readiness with which breathing air at all cold, or swallowing anything at all irritating, will excite cough. We have before remarked how this increased irritability of the inflamed air-tubes is commonly joined with the augmented irritation of their

secretion; but we do sometimes meet with cases in which the increased sensibility is purely nervous, unaccompanied by any secretion; and the cough is brought on by the slightest cold or irritating matter in the air. Even strong odours will sometimes cause it. These nervous coughs are to be treated chiefly by various remedies which diminish the sensibility of the nervous system, such as narcotics, or sometimes by those which excite stronger impressions in other parts, such as epispastics, and the application to heat.

3. Besides the sensibility of the bronchial membrane, another property connected with the nerves, muscular mobility, may be the source of some varieties of cough. We cannot here examine the circumstances under which a change of proportionate relation takes place between the action of the motory nerves in general, and the impressions which excite them. It is sufficient for us that the fact is well known, that in certain conditions or states of the system, an ordinary impression will excite inordinate motions; while in others the motions resulting from similar impressions will be imperfect, and below the natural amount. It is thus also with the motions of muscles concerned in coughing. They may be excessively mobile, so that the least irritation will set them a going: and like a clock without its pendulum, they continue their impetuous motions, until their strength has fairly run out. This is the *convulsive cough*, which we meet with in some hysterical and nervous subjects, and its convulsive character is the more evident from the fact, that it sometimes alternates with chorea or convulsive affections of other sets of muscles. The same uncontrollable character is, however, often communicated by a nervous temperament, or peculiar nervous affections, to coughs arising from common causes, which thus shake and exhaust the patient in an unusual degree, and require appropriate modifications of treatment to subdue them. *Hooping-cough* in its after stages is of this kind, and from my experience I should say that the shaking, uncontrollable nature of the cough is more characteristic of pertussis than the hooping, which is not always present, especially in adults. This leads me to consider on what hooping depends, and here again we find the use of our physiological divisions, which explain some other varieties of cough that are sometimes met with.

In the forcible expirations which constitute ordinary coughing, there is a simultaneous contraction of the air-tubes, and especially of the aperture of the glottis, through which the air is driven with the greatest force, in order to expel any irritating matter. Now the contraction of these tubes may be excessive, defective, or irregular, and this will occasion other varieties of cough. When this contraction is excessive, being also generally irregular, they give the *wheezing* character to the cough, so remarkable in asthmatic subjects. Not that wheezing cough always depends on contraction of the circular fibres, for other constrictions of the bronchi will also cause it: but if we listen to the chest of a nervous asthmatic, we may often hear, in the forcible expirations of a fit of coughing, sibilant and sonorous rhonchi, which are too transient to be produced by the thickening or secretion of the tubes. Where the irritability of the bronchi is great, their contraction may not, as usual, cease during the act of inspiration; and it is this spasmodic constriction, affecting particularly the upper part of the air-tube during the forcible inspiration which succeeds to coughing, that causes the *hooping* sound. This state of things happens chiefly in the irritable frames of children when affected with convulsive cough; and the violence and repetition of the expiratory effects of this cough occasion the back draught to be the more forcible, sonorous, and prolonged. If we listen to the chest of a child during a fit of hooping-cough, we are surprised to find how little sound we can hear there with all these noisy external efforts: in fact, the continued constriction of the bronchial tubes permits very little motion of air into and out of the tissue of the lungs. In the convulsive cough of adults, again, in which there is no hooping, the respiratory murmur of the long inspiration, or back draught, is pretty loud, whilst the succession of coughs here also consists more of external than of internal movement. In all these kinds of cough, antispasmodics will often give more relief than any other class of remedies.

But we may have an opposite condition of the circular fibres of the bronchi, a weakness or deficiency of action, a paralysis, so that they do not contract as usual during the expiratory efforts of coughing. This constitutes the *hollow* or *barking cough* which we sometimes hear in chronic bronchitis, and now and then in febrile and nervous affections. This cough is, as we shall presently find, accompanied with a difficulty of expectoration; hence it is sometimes very distressing, and particularly so when, as occasionally happens, it is combined with a mobility of the external muscles of respiration, rendering the cough convulsive and paroxysmal. The tearing and exhausting fits of this kind of cough are sometimes quite agonizing; and we may judge from the bloated, congested appearance of the lips and face, how much these fits impede the respiration and circulation, and how much they may thus tend to increase and perpetuate the diseased condition of the bronchial tubes. In some such cases, I have seen the terebinthinaceous medicines, with external counter irritation and occasional emetics, give most relief; but the treatment will depend on various circumstances, which I cannot enter into at present. Another modification of the nervous cough is the howling or shrieking cough; which depends on the addition of a vocal sound to the cough, the rima glottidis being spasmodically contracted to a vociferous degree.

4. Besides the sensitive and motory apparatus concerned in the act of coughing, we have the secretion of the air-tubes, which may also by its qualities modify the character of the cough. According to whether this secretion is present or not, the cough may be humid or dry; and according to the relation of the qualities of this secretion to the powers of expectoration, the cough may be loose or tight; and these varieties may be combined with the other species of cough, as those may with each other; and thus is produced the endless host of different kinds of cough that we meet with in practice. I have often found the preceding analysis useful in drawing attention to the predominant changes of vital property, as manifested by this symptom, and in thus distinguishing cases which require different modes of treatment.

The *expectoration* is another symptom of thoracic disease, which must be considered as the result of vital as well as physical properties, and therefore I include it under the head of general diagnosis, although it sometimes approaches in character to a physical sign. The word expectoration strictly means the *act of expelling* anything *from the chest;* but by a figure of speech it is also applied to the *matter so expelled.* We shall find that both the act and the matter of expectoration may present signs of the condition of the pectoral organs.

If we consider the structure of the bronchial tree, we shall perceive that natural breathing tends to prevent the accumulation of matters in its tubes, in spite of gravitation. The area of the smaller divisions of the bronchi is considerably greater than that of their trunks; and it may be represented as the divided base of a hollow cone or funnel, which is concentrated in the trunks, and lastly in the windpipe. Now the air, in the more sudden act of expiration, passes with greater rapidity and force as it converges into these trunks, and therefore tends to carry through them any superfluous matter that may be present on the bronchial surface. This will explain how the finer bronchial tubes of the most dependent parts of the lung are, in health, kept clear of any accumulation. Possibly the ciliary motions of the mucous membrane may, as MM. Purkinjie and Valentin have surmised, tend to the same effect. In confirmation of this notion, I may state that Dr. Sharpey has observed in the trachea of a puppy that the ciliary motion impelled a current towards the larynx.—*Cycl. of Anat. article* "*Cilia*," p. 632.

But it is the forcible acts of special expectoration, *hawking*, and coughing, that tend most effectually to clear the air-passages; and they do this by both increasing the force and fulness of the expiratory effort, and at the same time contracting the upper tubes and trachea, so that the air acts with greater force on any superfluous matter in them. The repeated closure of the glottis in coughing increases the expulsive effort by letting out the air in successive sudden jerks, which are more forcible than any continued act of expiration would be. We see this exemplified in cases where the operation of bronchotomy has been performed. The patient often cannot expectorate effectually as long as air can pass out from the artificial opening, and he is in danger of suffocation in consequence; but, on closing this during the act of coughing, the force of the air can be directed in the natural way against the accumulated matter. By attention to this particular, suffocation has been averted in more than one instance after this operation. In certain diseased conditions of the larynx, the patient cannot close the glottis; and hence also expectoration may be difficult, and the cough assumes a continuous unguidable character, which may be added as another variety to those enumerated before. This is what M. Trousseau calls a *belching* cough.

There is another element essential to the proper performance of the act of expectoration,—the capacity to make such a full inspiration as shall carry the air in beyond the accumulating matter, so that it may, on its forcible passage out again, carry this matter before it. Hence we see why weakness, which prevents a sufficient inspiratory effort,—or obstruction of the terminal and most expansible parts of the air-tubes, which renders this effort ineffectual,—may stop the act of expectoration, and by permitting the accumulation of matter in the air-tubes, may speedily conduce to a fatal result. Inability to expectorate is the immediate cause of death in many cases of various diseases; in fact, it is a part of the article of death itself; and when we hear the rattle in the throat of the dying, it is the sign of the accumulating barrier which is shutting out the breath of life. Sometimes, even at this stage, there are sensibilities enough in the system to feel the force of a stimulant which may excite the sinking powers to another struggle; expectoration is once more accomplished, and breath once more renewed; and where there is no irrecoverable alteration of structure, this act of expectoration may in some few instances turn the balance in favour of recovery. How important, then, is it to study the act of expectoration, and to acquaint ourselves with those means that they may excite or promote it! I dare say that most practitioners have seen instances in which a patient has been snatched from the jaws of death by the timely administration of a diffusible stimulus, such as a warm aromatic draught, with carbonate of ammonia, together with such a change of posture and other circumstances as might most favour the expulsion of the matter that was suffocating him. A great deal may be done in less urgent cases by attention to the posture of the patient. In most instances the act of expectoration is easiest in that posture in which the respiration is most free, which is commonly the semi-erect posture; but I have known some patients expectorate more freely when lying on one side; and I remember a phthisical patient who really appeared to be several times saved from suffocation by alternating his posture from lying down to sitting up in a particular manner, suggested by a knowledge of the condition of the lungs in that case. When this expedient was neglected, the patient was so shaken with frightful fits of fruitless cough, and so oppressed with the accumulating matter which they could not expel, that speedy suffocation seemed inevitable. In some cases, the act of expectoration may be favoured by another kind of action, in which the expiratory muscles are concerned, that of vomiting; and we shall find hereafter, that some emetics may exert an influence of an important nature on the bronchial tubes, besides this mechanical one.

The *matter of expectoration* frequently furnishes us with very instructive signs. It is the product of diseased action, and in its physical or chemical qualities it may inform us somewhat of the nature of that action, of the condition, and sometimes of the position of the part from which it comes. As, however, we have seen that the effort of expectoration is sometimes unsuccessful, so there may be no matter of expectoration to judge of; and besides this, most children and some adults cannot spit out what they expectorate, but swallow it.

The basis of expectoration generally is the secretion of the mucous membrane of the air-tubes. This is naturally a transparent, colourless, slightly glutinous liquid, like thin mucilage. The chief animal matter which it contains is that called mucus, which seems to be a sort of imperfectly coagulated albumen ; and the varieties of sputa presented by disease commonly depend on an unnatural condition or quantity of this animal matter. There is also saline matter, which may vary in quantity, and so may the proportion of water. From the recent experiments of Dr. Golding Bird, as well as those of Dr. Babington and Mr. Brett, it would seem that the condition of the animal matter in the expectoration in great measure depends on the proportion of saline matter with which it is combined ; this being abundant in transparent and viscid expectoration, defective in the opaque kind, with little viscidity, and least of all in that which is absolutely purulent. Dr. Babington found that on mixing pus with a solution of common salt, after a time it became converted into a nearly semi-transparent viscid mass like mucus. Dr. G. Bird rendered the physical and chemical resemblance perfect by adding a little soda, and then passing a current of carbonic acid gas through it. (Guy's Hospital Rep. No. 6.) Mr. Brett, in a valuable communication to the medical section of the British Association in 1837, stated that he found the saline matter of transparent viscid expectoration to amount to from 20 to 33 per cent. of its solid matter, whilst that of the opaque mucus of chronic bronchitis was from 16 to 23, and the purulent expectoration of the last stage of phthisis was only from 9 to 10 per cent. These researches confirm the opinion that I have long held, that the difference between mucus and albumen seems to consist in their physical condition, rather than in their chemical constitution.

If microscopic appearances are to be depended on, the secretion of mucous membranes is rendered opaque by the scales or epithelic nuclei which are thrown off like the scurf from the skin. These are present in all mucus, but abound chiefly in that which is opaque and dense. According to Henle, the globules of mucus, as well as of pus, are the nuclei of the cells of the membrane, and are essentially the same in nature as the fibrinous globules in the blood. As this view is not yet generally received, I think it advisable to adhere to more common distinctions.

Mucus is a transparent glutinous matter, not coagulable by heat, as liquid albumen is, and not solid and opaque like coagulated albumen; but on ultimate analysis it is not found to differ from this principle. When, therefore, we see expectorated matter opaque and solid, or liquid and coagulable by heat, it loses the only distinguishing characters of mucus, and is strictly albuminous. For this reason, I propose the following general classification of expectorated matter:—

1. *Mucous*, more or less transparent and viscid.
2. *Albuminous*, opaque without viscidity.
3. *Watery*, thin and transparent.
4. *Compound*, composed of combinations of the preceding kinds.

1. *Mucous* expectoration is that most like the natural secretion, being transparent, and more or less viscid. It is the general result of simple acute inflammation of the mucous lining of the air-tubes, in which case it is increased in quantity, and particularly in viscidity; in fact, the glutinous character of the sputa, and the tenacity with which they stick together, and to the containing vessel, or fall out in a ropy mass, was described by Andral, and I think correctly, to be a mark, and, in some degree a measure, of acute bronchitis. From the researches just alluded to, it would appear that the viscidity of these sputa depends on their quantity of mucus, which is albumen combined with saline matter, to which are sometimes added free uncoagulated albumen. In the most intense forms of inflammation, and where the disease occupies the finer tubes, to the glutinous character of the mucus is added a frothiness, arising from the mixture of those air-bubbles in the tubes, which, in their breaking, cause the mucous and submucous rhonchi. But the most intense bronchitis is that accompanying inflammation of the parenchyma; here we have the most viscid form of sputum, through which air driven produces the crepitant rhonchus; and the blood in the distended vessels of the engorged parenchyma communicating a little colouring matter to it, gives it that reddish or rusty tinge which is so characteristic of the sputa of peripneumony. The transparent or semi-

transparent condition of these viscid sputa distinguishes them from the albuminous kind, into which, however, they pass in the advanced stages of all the more inflammatory affections of the bronchial membrane. The mucous expectoration has commonly a saltish taste, and with its saline matter is probably connected its irritating quality, so marked in the early stage of bronchitis.

2. The varieties of *albuminous* expectoration are pretty numerous, for I comprehend under this head the opaque kinds of sputa which have no remarkable viscidity, such as the purulent expectoration of chronic bronchitis, the fibrinous or polypous sputa of plastic bronchitis, and the more compound combinations of these with caseous and other matters, which are voided in the advanced stages of pulmonary phthisis. This class of sputa denotes an error of secretion further than the mucous from the natural standard; there being a defective proportion of saline matters, as well as an excess of albumen; but their production generally announces a decline of inflammation from its most acute form. Probably, the very throwing off of so considerable a mass of animal matter is the means of relieving to a certain extent the inflamed vessels; for we frequently find the purulent or polypous expectoration in intense bronchitis attended by a remarkable diminution in the signs of local and general excitement. This remark has been also made by Dr. Stokes. But such an expectoration ceases to be a favourable sign when it *continues*, either with undiminished irritation, or with proofs of general weakness; for then a change is implied, either in the structure, or in the habitual action of the membrane, which, secreting pus instead of mucus, goes beyond the mere removal of a temporary congestion, and proves of itself a cause of irritation and exhaustion. Much has been written about the modes of distinguishing pus from mucus in the expectoration. On these formerly the diagnosis of pulmonary phthisis was supposed to depend. These tests are now little attended to; not only because it is well known that pus may be produced without any ulceration or consumption of the lung, but also because these distinctions cannot be complete between matters that pass by insensible gradations into each other. The chemical composition of pus resembles that of the globules of the blood, and differs from mucus in containing a notable quantity of iron. According to Mr. Gulliver, the structure of the pus globule is peculiar, being a vesicle larger than the blood globule, and containing one or more nuclei.

3. *Watery* expectoration is that kind in which a liquid of only slightly glutinous quality is coughed up in greater or less abundance. This appears to contain very little animal matter, and to be rather a diluted mucus than to have in it anything peculiar. It is often covered with a froth, particularly when it is coughed up with much effort. This secretion I regard as the result of irritation with a relaxed state of the vessels, rather than of inflammation; but it may occur as a consequence of this lesion, as well as of congestion or obstruction to the circulation of the blood in the lungs. It is the expectoration of what is called humid asthma and pituitous catarrh. Some persons of a relaxed habit have it during a common cold, or any form of bronchial inflammation. It sometimes tastes more salt than usual, and in this case it commonly causes a more incessant teasing cough.

4. Under the head *compound* expectoration, I class various combinations of the preceding kinds, which we meet with in almost every form of pectoral disease. They are either products of different parts, in distinct pathological conditions, although coughed up at the same time; or they may in some cases proceed from the same part in an intermediate pathological state, and capable of secreting different kinds of matter. An example of the latter is the opaque or mucopurulent expectoration of the latter stages of bronchitis, in which the opacity and colour of albuminous matter is apparent, whilst it is held together by a mucus of some tenacity. In the *concocted* sputa of declining acute bronchitis, the mucus predominates; whilst the loose albuminous matter is more abundant where the inflammation tends to pass into a chronic state. The sputa of chronic bronchitis, and in fact of most chronic diseases of the lungs and air-tubes, are almost always more or less mixed; for it generally happens that the different parts of the membranes and tissues are variously affected; and when, as in the advancing stages of phthisis, there is structural lesion or destruction of parts, there is the greater reason for a more heterogeneous kind of expectoration. In these cases, however, the albuminous kinds mostly predominate, in the form of muco-purulent, purulent, caseous, or tuberculous matter, and coagulable or fibrinous lymph, occasionally tinged or mixed with the colouring matter of the blood; these constitute the bulk of the expectoration of the consumptive. In catarrhal diseases of a chronic kind, we very commonly see very opposite forms of sputa expectorated together. Thus in a spitting-dish full of thin,

frothy, watery expectoration, we often find portions of tough and almost solid semi-transparent mucus, as if some parts of the tubes were throwing off the water, and others the animal matter, in a separate form. When we come to the subject of catarrh, we shall find that these opposite products do not imply an equally opposite pathological condition. After hæmoptysis, it is very common to see fibrinous concretions, together with purulent and mucous matter, all more or less tinged with blood. In other affections, it is not uncommon to see the sputa streaked with blood; and this sign is of less importance when the cough is violent, because it may then merely proceed from a slight abrasion caused by the force of this mechanical action. When, however, there is often blood present, without much force of cough, and especially if there be pus with it, we may suspect the presence of ulceration in some part of the air-passages. The colouring matter of the blood, in an altered state, may also be combined with other forms of sputa. Thus, in scorbutic persons affected with humid catarrh or bronchitis, the expectoration is a thin reddish-brown liquid, like prune juice or diluted treacle; and in the last stages of pulmonary disease, the colouring matter, from the final pulmonary congestion which precedes death, is seen in the dirty reddish-brown or greenish tinge of the purilaginous sputa.

It is plain, then, that the matter of expectoration will often inform us of the pathological condition of the lungs and their tubes; and its quantity or quality may sometimes suggest proper remedies. In some cases we may learn other things from it. Thus, when in consumption, tubercular matter, with portions of pulmonary tissue, are expectorated, the conclusion is obvious. We also sometimes see the expectoration present physical signs of the state of the interior, by its containing albuminous or compound matter, moulded into the shape of the tubes or cavities from which it comes. The large rounded or ragged flocculent muco-purulent sputa of advanced phthisis are often such as could only accumulate in a cavity; and the tubular or vermicular albuminous matter which is coughed up in the plastic kind of bronchitis, sufficiently explains whence it comes, by its being an exact mould or cast of the bronchial tubes, sometimes in an arborescent form, from several of their divisions.

I must not omit to notice a test, which is erroneously used to determine the nature and source of sputa,—whether they float or sink in water. Now the floating of a sputum merely depends on the number of air-bubbles retained in it; and although pus alone, or tuberculous matter alone, will not retain these bubbles, yet a small addition of tenacious mucus will enable them to do so. Again, although the sputa formed in ulcerous cavities are less likely to contain air and to float than those formed in the tubes, yet we not unfrequently find the concocted expectoration of acute bronchitis, which is formed exclusively in the large tubes, sink in water; whilst the mixed product of a vomica and the adjoining tubes, which has been churned together with air, floats. This *hydrostatic* test of expectoration is then a very inconclusive one; but it may be useful in sometimes causing a rough separation of the albuminous matters from those of a more viscid mucous kind.

Other details regarding the matter of expectoration might be added; but enough has been said to illustrate how it may prove useful in diagnosis and practice. From this it will appear how much more valuable its indications may become when conjoined with the physical signs, by which we may often detect the position, and measure the amount of the local disease, of which the matter expectorated is the product. We shall find many exemplifications of this position hereafter.

The only other morbid phenomenon connected specially with the modified vital properties of the organs of respiration is *pain*. We know that pain may arise either from an excessive impression on the nerves of sensation, or from an excessive sensibility of these nerves, to which common circumstances of position, motion, &c., then become painful. The latter is the more common cause of pain in internal diseases, but it is not unusual to find them combined, as when a tumour, or effused matter, presses on or stretches parts morbidly sensible. The most common causes of pain are inflammation, and those kinds of vascular excitement that are allied to it; this vascular excitement is generally attended, in the first instance at least, with an exaltation of the nervous function. But the nervous function may be *primarily* excited; and although the increased sensibility thus produced seldom lasts long without more or less stirring up the function of the vessels also, yet we may for a time have pains purely nervous, such as pleurodyne and pectoral neuralgia. Further, as inflammation is not the only cause of pain, so the pain present in inflammation is by no means an index of the extent of the inflammation, nor even of its situation. Most extensive inflammations have been known to occur, not only in the parenchyma of the lungs, but in the bronchial membrane and pleura also, without producing any pain; and it frequently happens in phthisis, that the pains chiefly complained of are low down in the sides, when the disease is almost entirely in the upper lobes of the lungs. So likewise in bronchitis and pneumonia, the pain is often confined to the sternal, lateral, or scapular regions, whilst the disease occupies other parts.

There are, nevertheless, some general characters with regard to pain, which may render it useful as a symptom of disease of the chest. It is commonly remarked, that the pain of parenchymatous and bronchial inflammations is dull and diffused, whilst that of inflammation involving the serous and fibrous membranes of the pleura and pericardium is of a sharp lancinating character. This is generally, but not constantly, true; and we may find it explained by the circumstance that the par vagum, which supplies the bronchi and lungs, is by no means so sensitive a nerve as the spinal intercostals, which are distributed on the pleura and pericardium. For this reason, too, there is more apt to be acute pain when the costal pleura, or the coverings of the great vessels, are inflamed, than when the pulmonary pleura is the chief seat of disease. A further distinction in the variations of these several kinds of pain will confirm the opinion, that they belong to different orders of sensibility. The dull, heavy, or aching pain of bronchitis, or pneumonia, is generally pretty constant, although it is increased by full inspiration, exertion, or the breathing of cold air; yet even then it gives the feeling of soreness under the sternum, rather than of severe pain. It often resembles the pain of dyspepsia, which is probably seated in a branch of the same nerve, and is also usually referred to the sternum. The degrees of pleuritic pain, on the other hand, are sudden, extreme, and intolerable. If it be not felt in ordinary breathing, a long breath, or a cough, just sufficient to bring the membranes to a requisite degree of tension, causes that sharp stitch of the side—that sudden catch of the breath, that has been considered so characteristic of pleurisy. When it is constant, the patient is obliged to hold his side to diminish its severity, by restraining the motions; and thus placed in opposition to the sensation which prompts the act of respiration, this sharp pain may cause such a voluntary restraint of these acts as to bring the patient to the verge of asphyxia. It is under these circumstances that the breathing becomes partial, as formerly described; and patients whom pain constrains to breathe only with the diaphragm, or with one side, will perform this supplementary respiration so well, that they are completely free from pain, although the inflammation is as acute and the membranes as tender as ever. Sometimes we may detect the latent tenderness by pressing between the ribs of the affected side; but we are more likely to succeed if we restrain the supplementary respiration by pressing on the abdomen, or on the healthy side, and then desire the patient to cough, or to take a sudden long breath. If there be any exalted sensibility, or tenderness, it is pretty sure to be discovered by this means; and I have several times met with patients who denied having any uneasiness, or tenderness, yet they winced at the pain developed in this way.

As a general rule, (not, however, without some exceptions) we may consider a fixed permanent pain, or a permanent tendernesss, which depends on the same pathological cause, to be an indication of inflammation, or congestion, or some analogous condition of the vessels; and when present it deserves attention, not only as an object of treatment on its own account, but also as an index, which, together with the pulse, cough, fever, and other general symptoms, shows the increase and diminution of the complaint, and the effects of remedies, even before these become manifest from the physical signs. Still, if we trust to it alone, it will negatively deceive us in those numerous cases of extensive disease in which it is absent, or scarcely complained of; and it will positively deceive us in those cases in which modified nervous sensibility—a mere neurosis—is the only or the chief disorder.

We come now to examine shortly the nature and value of the symptoms which diseases of the organs of respiration develope in other functions.

Physiology points out the close relations which subsist between the organs of respiration and those of circulation, and prepares us to expect that disease in the former would disturb the latter, and develope symptoms in the function of circulation. Accordingly, we find such symptoms in the state of the *arterial pulse*, and in that of the *superficial capillary* and *venous* parts of the circulation.

The *pulse* has for ages been relied on as a guide in the diagnosis and treatment of all diseases; but those who have had much experience know how fallacious it sometimes proves; and those who have had little experience must acknowledge, that it is very difficult to distinguish the varieties of the pulse from one another. Now it appears to me, that some of the fallacies and difficulties connected with the pulse as a sign, arise from our studying it too empirically—from our not rationally considering those elements on which its varieties depend, and a knowledge of which would enable us to understand and to foresee the circumstances which are capable of producing these varieties. As the subject of the action of the heart will more fully come under our notice in the latter part of this work, I shall now only add a brief analysis of the nature and varieties of the pulse of arteries.

The arterial pulse is caused by the jets of blood thrown at certain intervals of time into the arteries by the contractions of the ventricles of the heart. The motion originates exclusively in these contractions, although it may be modified by the blood which is moved, and by the tubes which convey it. Now here are three elements:—1, the heart; 2, the blood; and 3, the arteries;—and variations in the condition or action of each of these cause varieties in the arterial pulse. Let us consider a few of these variations.

1. Without noticing the modifications in the action of the heart resulting from disease of that organ—a subject to be considered hereafter—it is plain, that if the other elements be equal, the strength and frequency, or rhythm, of the contractions of the left ventricle of the heart, will determine the *strength* and *frequency* of the arterial pulse. But the contractions may have another quality—that of abruptness: being rather brisk and short than strong and complete, they communicate to the pulse that character which is called *sharp*. Now what property in the heart gives it this abruptness of contraction? What but an extreme irritability? There is sometimes this irritability in inflammations and fevers; but we find it also in conditions of mere nervous irritation, of which it is more distinctive. And it is when these co-exist with inflammation or fever, that the pulse presents a sharpness, in addition to other qualities more peculiar to inflammation. In sthenic irritations, or those connected with fulness and tone of the vascular system, which may tend either to acute inflammation or to active hæmorrhage or other discharge, the heart's contractions are strong as

well as sharp; and so is the pulse. In these cases, although the irritation originally were local, it has now reached the centre of the circulation, and thence, distributed through the whole system, becomes general.

2. There can be no doubt that the blood in the heart and vessels determines by its *quantity* the character of the arterial pulse: very possibly it does so by its *quality* likewise; but this is not so easy to prove. The fulness and strength of the pulse in the arteries depend materially on the quantity of blood in them; and when the pulse is frequent as well as full, there is the greater proof of plethora, inasmuch as it shows that there is a considerable jet thrown into the arteries at each contraction, notwithstanding that the contractions are so frequently repeated. But there may be a full system of blood-vessels without a large or strong pulse—as when the heart is acting feebly or faintly; and where its irritability is lowered, such a mode of action may be actually caused by the congestion or distension which for a time oppresses the function until it is roused into action. Under these circumstances, blood-letting will often increase the fulness and strength of the pulse. The opposite condition—a defective quantity of blood—will modify the pulse differently according to the state of the other elements, the action of the heart, and the arterial tubes. When the irritability of the heart is reduced, together with the quantity of blood, the pulse will become softer, weaker, and less frequent. But it frequently happens, especially in nervous temperaments, or where the depletion has been carried to excess, that the diminution of the blood is accompanied with an augmented irritability of the heart, and the pulse becomes not only quicker, but sharper than usual; and the effect of the abrupt jets into a small bulk of blood, contained in imperfectly distended tubes, is to give to the pulse that jerking or bounding character,—as if a mere ball of liquid were suddenly shot through the empty tube,—which is so remarkable in the irritation of inanition and chlorosis.

3. But we cannot fully understand the variations of the pulse without attending to the properties of the tubes in which it is felt. If the arteries were tubes of an unyielding or an unvariable character, then the pulse in them would more uniformly represent that of the heart, which would be transmitted through them unmodified. But we know that they are not so: they possess properties of *elasticity* and *tonicity*, which vary according to circumstances, and which

modify the pulses from the heart, by changing the size of the tubes, and the yielding or the resisting capacity of their walls. It is obvious that the impulse of a jet of blood must be differently transmitted by vessels when they are large and yielding, and when they are contracted and tense: in the first case the pulse would be *soft* and *full*; in the latter, *hard* and *small*. We know but imperfectly what are the circumstances which affect the tonicity and elasticity of the arterial coats, and thereby the pulse: further experiments are wanted to elucidate them; but the following are pretty well ascertained, and they should not be forgotten in estimating the signs of the pulse. Cold causes the arteries to contract, and therefore renders the pulse smaller. A cold lotion will often diminish the fulness and throbbing of the arteries of an inflamed part; and I have seen the same effect of cold more strikingly produced in the large arteries. In the experiments on the sounds of the heart which I carried on in February 1835, I repeatedly observed that when the aorta of an ass, recently killed, was plunged into cold water, it contracted, so as not to permit the introduction even of the little finger, and its coats acquired an increased thickness and rigidity: the pulmonary artery did not contract near so much. The circumstance of temperature must, therefore, be taken into account in judging of the pulse; for cold may render the pulse of an artery small and hard, or if severe, small and weak, when the action of the heart and the condition of the system would give it the reverse qualities. Heat, on the other hand, within certain limits, tends to diminish the tonic contraction of the arteries; so that under its influence they receive more strongly and fully the pulse from the heart. We know how warmth restored to a limb makes it throb with these expanded pulses.

But there is another circumstance that may modify these actions of heat and cold on the pulse, besides proving by itself a cause of modification—the condition of the capillary circulation. When this is not free, the artery will be more distended, and therefore the pulse harder and stronger than usual; and thus in fevers, where the surface is pale and constricted in the cold stage, and dry and unrelaxed in the hot stage, the pulse often preserves through these changes of temperature a hardness and strength which would be much more varied were the capillary vessels free and exhaling their usual excretion, and which are actually diminished under the influence of a warm bath or temporary moisture of the skin, although the fever still continue. Again, whatever view we take of the nature of inflammation, we cannot, in the present state of pathological knowledge, doubt that the circulation through the inflamed vessels is to a certain degree obstructed; whilst, either as a consequence of this, or from some co-operating influence, the vessels leading to the part become dilated, and being thus more open than others to the pulse-wave from the heart, which their distended coats cannot *temper* as usual, they become the seat of that throbbing hard pulse, that has been mistaken for increased action of the vessels themselves. There are many other variations in the pulse explicable on these principles, but to consider them further in detail would be out of place here.

There is, however, one more cause of variety connected with the arteries so frequently occurring, that it must not be overlooked,—I mean the difference in the arteries of different individuals. Without any adequate difference in the action of the heart, in the quantity of blood, or in the temperature, we find a very remarkable variety in the character of the pulse in different healthy individuals; and the same difference extends to the modifications of disease. Some have always a soft large pulse; others a small feeble one: in others, it is small and hard: others, again, have habitually a hard strong pulse, which scarcely becomes soft under any circumstances. The first depends on the arteries being large, with thin elastic coats. The small feeble pulse may result from their small size and thin coats: this is common in females, and may co-exist with inordinate action of the heart. The hard wiry pulse is connected with small arteries with rigid coats; and the same rigidity or deficiency of elasticity in the coats of arteries of larger size gives that unvarying hardness and strength to the pulse, which we so often meet with in old people, and which renders it so uncertain a sign in these cases. We may often, in the radial artery, feel the permanent thickening and hardness of its coats, which thus, like a tube of glass or metal, rigidly transmit the heart's pulses, without tempering them by any yielding or spring. With these peculiarities of pulse there are often connected characters of constitution or temperament, and proclivities to disease or health, which are of great importance in guiding us in practice.

Besides the general causes which modify the pulse, which we have now briefly considered, there are some specially connected with diseases of the pectoral organs. Those arising from diseases of the apparatus of the circulation will be treated of hereafter. But severe affections of the respiratory organs also sometimes signally modify the pulse, and that in a manner which may tend to confuse its indications. We know how closely the heart is linked with the lungs; by the circulation even more closely than by mere position: for the lungs may be said to lie between the two compartments of the heart, and any considerable obstruction in the lungs will derange the usual relations of these compartments. There is then a distension or over-stimulation of the right side of the heart; while the left, receiving a diminished quantity of blood from the lungs, and that not thoroughly aërated, is less excited than usual, and may give to the arterial pulse a character of weakness and smallness that by no means represents the condition of the whole vascular system, and which often is remarkably contrasted with the action of the right side of the heart, as felt or heard under the sternum. These varieties are produced by any of those affections of the chest which infringe far on the respiratory function. These are more commonly those of the bronchial and parenchymatous kind, which have accordingly been described to be accompanied by a softer and weaker pulse than those affecting the serous membranes. But we may have a pleurisy also with a small weak pulse, when the effusion or pain is such as to interfere largely with the function of the lungs. Neither is it to be supposed that the pulse in severe pneumonia or bronchitis is always weak, even when these affections infringe considerably on the function of the lungs. Even under asphyxiating influences the left ventricle may sometimes become excited, together with the right, and give a sharpness to the pulse, which, combined with the arterial tension of fever, may be readily mistaken for hardness and strength. But this character is seldom permanent; and we generally find in all diseases, when the function of respiration is much impaired, that the pulse soon loses its body and strength. These considerations suggest the expediency of examining the state of the circulation not only by the arterial pulse, but also by the pulsations of the heart itself, and by the conditions of the veins and capillaries.

Under the circumstances just mentioned, when the indications of the arterial pulse are most variable and deceptive, we may often find useful signs in the condition of the *venous* and *capillary* part of the circulation. The distension of the more superficial venous trunks, especially the jugulars, in which a double pulsation often shows also the retropulsive action of an over-distended right ventricle; the fulness of the capillaries of the lips, tongue, throat, cheeks, eyelids, nails, and other parts, at first having a florid and flushed appearance, but afterwards, as the respiration becomes more injured, assuming a purple or livid hue,—are signs of great practical importance, and of a constancy more approaching to that of the physical signs. They do not, however, present themselves in the early and more tractable stages of disease, and they are always less distinct in pallid persons with small superficial vessels.

Intimately connected with the state of the circulation is the symptom of *general fever*, or *increased heat*, which attends many diseases of the chest. It depends on increased force and rapidity of the circulation, with diminished perspiration. When the perspiration is restored, the heat always falls. This exhalation of fluid not only lowers the temperature by its physical agency of evaporation, but being in itself a sign of a relaxing of the superficial vessels, it implies an abatement of the vital irritation. In the more transient forms of fever, such as the intermittent and hectic, the profuse perspiration sometimes reduces the animal heat to below the natural standard, just as the circulation is proportionably enfeebled; and the same chilling influence is illustrated by the cold sweats which succeed to temporary and irregular excitement. There are degrees of vascular irritation in which the increased heat of skin is partial, and determined by the structure of particular parts. Thus in the asthenic excitement of hectic fever, the heat is most felt in the palms of the hands and soles of the feet, because the circulation is not strong enough to drive the perspirable excretion through the thick cuticle of those parts, which become consequently dry and hot. The same thickness of cuticle, however, when once imbued with perspiration, often keeps these parts soft and moist, when there is no sensible perspiration on other parts. Not unfrequently the unequal state of the circulation is exhibited in febrile and inflammatory disorders by the heat of the abdomen, back, chest, or head, whilst the extremities are cooler than usual; and occasionally the same locally increased action is manifested by partial sweats, which prevent the increase of heat, and tend to reduce the excitement. I have known a patient with pleurisy perspire profusely only from the affected side for several days; and nothing is more common, in slight abdominal inflammations, than to find the pungent heat of the belly relieved by a perspiration equally confined to that part. We practise on the same principle, in applying to irritated or inflamed parts poultices, fomentations, and partial baths, which tend to bring the skin and superficial vessels to the same relaxed and expanded state which they have in a perspiring part.

I think, then, that it will be plain that heat of skin must be an uncertain symptom; for it depends on a condition of the superficial circulation that is by no means constantly associated with disease of the internal organs. When present, it may as much result from a general cause—an idiopathic fever—as from a local inflammation; and cases are not uncommon in which severe, and even fatal, visceral inflammations are attended through a great part of their course by free perspiration: nay, the same may be said of some fevers which are called idiopathic. Still the heat and condition of the skin become valuable guides, when taken in conjunction with other signs, inasmuch as they indicate the constitutional disturbance, which is an important part of the disease, and which is sometimes as much to be considered in the treatment as the local disease which has excited it. So, likewise, when the presence of a disease has been established by other signs, the condition of the skin may prove a measure of its increase or diminution more delicate and sooner appreciable than can be found in the physical signs. Thus an increased heat of skin, coming on during a bronchial or pulmonary inflammation, either indicates an increase of that inflammation, or the addition of some abdominal or other irritation, which tends to aggravate the condition of the patient. So, too, perspiration breaking out in the hitherto dry and hot skin of a pleuritic patient, occasionally does prove *critical*, whether that word be applied to the excretion as a cause or as a sign of the amendment.

When there is disorder of the circulation, especially of a febrile kind, we may well expect alteration of the *secretions*, which are so intimately connected with it: hence we find the urine is scanty and high-coloured, and the secretions of the liver and intestines variously deranged. As a natural consequence, too, there will be disorder of the digestive and nutritive function: the tongue will be furred, or florid; the appetite will fail; the stomach will cease to digest; thirst will torment; the blood, no longer fed with chyle, will not duly nourish the textures, nor support the functions; the strength will fail; absorption continuing active, if time permit, emaciation will ensue; and various complications of these disturbances may differently modify the character of diseases of the chest. The sensorial functions, too, may be deranged, either in consequence of the secondary visceral disturbances, or more rarely by a more direct influence of the imperfect respiration on the brain and nervous system. A knowledge of the causes of asphyxia suggests also how certain states of the nervous system may tend to develope disease of the lungs. Thus insensibility, or coma, causes imperfect respiration, and consequently congestion of the lungs; and, as we shall hereafter see, a long-continued congestion of the lungs only requires the addition of vascular reaction to convert it into inflammation. Persons rarely recover from an asphyxiated state, without suffering more or less from the injury which it leaves in the vessels of the lungs; and not a few who have been recovered from suspended animation have sunk under the pneumonia, or bronchitis, which supervened.

I have thus rapidly glanced at some of the pathological relations of the organs of respiration to other functions, to give the reader an opportunity of considering rationally the nature and value of general symptoms. We have said enough of those more nearly related to the chest,—dyspnœa, cough, pain, and signs of the circulation, with its concomitant, temperature: although often equivocal, yet when strongly marked, they sometimes assist us in diagnosis. Now it is just the reverse with the symptoms arising out of disorder of the other functions. The altered secretions of the kidneys, the liver, and the intestines, cannot inform us of the nature or presence of a disease of the chest; and still less will gastric derangement or sensorial disturbance. Nay, not only will they not direct, but they tend essentially to blind us to the presence of pectoral disease; for they set up prominent symptoms of a new character, that may take the attention entirely from the real source of disease, and fix it on the brain, the liver, the stomach, or intestines, the affections of which are only secondary, and often trivial. How often do we find a peripneumony, or a bronchitis, disguised by delirium or stupor, or by vomiting, a loaded tongue, or diarrhœa! How often a pleurisy, masked by a jaundiced skin, a tender right hypochondrium, and clay-coloured fæces, or by a lumbago, or a nephralgia! How often tubercular consumption, obscured by sundry bilious, dyspeptic, or nervous symptoms! It will be happy for the practitioner, (for his credit at least, if not always for the success of his practice,) if he detect the enemy through its false colours ere it triumph, and before the scalpel shall proclaim his error. The physical signs will enable him to do this, and again I commend them to the best attention of the student. On taking a rational review of the general symptoms, in comparison with the physical signs, I think all must come to the conclusion to which my reason and experience have long brought me,—that, as diagnostic means, the general symptoms fall far short of the physical signs.

But it is not to be supposed that because the general symptoms are often comparatively of little aid in diagnosis, we are to neglect the study of them. They are almost always of great importance in prognosis and practice. The physical signs more surely show how the pectoral organs suffer; but having discovered this, to the general symptoms we must look for how the system suffers; and as the system often closely sympathizes with the injured organ, we may through them often watch the first turns of the disease before the change in the organ becomes physically appreciable. In the general symptoms we seek for those *critical* phenomena, which, although sometimes deceptive, yet generally announce the tendency of the disease to one or other mode of termination. In them we study the vital forces and properties with which nature works, and the signs of what nature can do; and in our methods of treatment these become the standards to which we direct, and by which we modify, our remedies. When we treat a patient with peripneumony or catarrh, we do not apply our remedies merely to the local lesions, inflamed vessels, or a discharging membrane; we study the system at large, we examine other functions through the general symptoms, and we direct our treatment with due reference to indications from all these several sources. We see, then, that the mere stethoscopist is but ill fitted to practise medicine. He may justly boast of his skill in diagnosis: his place of triumph will be the dissecting-room, where he can show the lesions that he had detected; but his practice at the bed-side will be unsuccessful in proportion as local lesions vary in their general relations, and in the conditions of the constitution, or of other functions that may accompany them. The judicious physician will not omit to study the condition of the vital properties, which are exhibited in the general symptoms, as well as the local physical changes which have been already produced; and whilst he chiefly confides in the physical signs to indicate and measure the present local lesions, he carefully watches in the general symptoms the tendencies of those properties and functions which are capable of increasing or modifying these lesions, and are equally liable to be affected by them. The general symptoms being less intelligible and certain than the physical signs, need more experience to enable us to appreciate them; but we have found that even these also may be *rationally* studied, and may derive a light from a knowledge of physiology and of the physical signs, which experience alone could never throw on them.

PART II.

THE PATHOLOGY AND PHYSICAL SIGNS OF SPECIAL DISEASES OF THE LUNGS AND PLEURA.

HITHERTO we have considered physical signs only with relation to the natural or physiological state, and to the general pathology of the thoracic organs: it now becomes our task to study the forms or characters that individual diseases present to the auscultator. To understand the physical signs of a disease, it is quite obvious that we must be acquainted with its pathological characters, and especially the physical changes of function or structure which the disease produces in a part; and I view it as not among the least advantages of physical examination, that it directs our attention through a confusing crowd of uncertain and equivocal symptoms of general derangement, to that substantial and primary lesion, which, if not the starting point of all, is that against which our practical efforts should be chiefly directed.

In this elementary treatise, the object will not be to enter into minute details of pathology and morbid anatomy; but, as far as my own observations and those of others will enable me, to explain the general physical nature of the changes which, in individual diseases, so modify the conditions of the thoracic organs, as to produce signs available as means of diagnosis, and as guides in practice. With this pathological exposition will be occasionally blended short accounts of the leading general symptoms, which, although often equivocal or deceptive in themselves, yet, taken in conjunction with the physical signs, greatly contribute to the useful and practical knowledge of disease. The text will generally be confined to pathology, properly so called, but I shall not omit to append, in form of notes, a description of the general morbid appearances found after death, by a comparison of which with the physical signs during life, the real nature of a disease is to be known.

CHAP. I.

DISEASES OF THE AIR-TUBES.

Bronchitis.

THE pathological cause of *bronchitis, acute mucous* or *pulmonary catarrh*, is an inflammation and altered secretion of the mucous membrane of the bronchia. There are several varieties of this disease, arising from its extent, or from the state of constitution or complications with which it is associated; but as they pass insensibly into each other, and as the physical signs of all are frequently combined in one, I shall comprehend in this section their general description, distinguishing only the acute and chronic diseases.

1. *Acute Bronchitis.*—Inflammation of the mucous membrane of the bronchi, like that of the nasal passages in coryza, at first causes effusion into the interstices of the mucous and submucous tissues: the result of this is tumefaction and partial obstruction of their caliber. This partial obstruction, or constriction, occurring in individual points, so modifies the passage of air through the bronchial tubes, that it produces sound after the manner of instruments of music. If the whole periphery of a portion of a tube be tumefied, the constriction is circular, and the air passing through it produces a whistling sound. This constitutes the *sibilant* or *whistling rhonchus.* If the tumefaction be unequal, so that the constricted portion preserves a flattened aperture, then a sound is produced after the manner of reed instruments, or rather of the horn or trumpet, by the rapid alternate compression and dilatation of the air passing between two vibrating laminæ or surfaces. Such, I conceive, is the rationale of the *sonorous rhonchus*, the *droning* or *humming* breath-sound. The extent of the constriction, its situation, and the secretion lubricating the tube, will variously

modify the note and tone. The larger bronchial tubes alone can produce deep or bass notes; but it is plain that they may also yield high ones. When a deep sonorous rhonchus is produced in a bronchus near the surface of the lung, it communicates a slight vibration to the corresponding wall of the chest, which may be felt by the hand. This mechanical vibration is often perceived internally by the patient himself, although he do not hear the sound that produces it. As the inflammation attacks the larger bronchial ramifications first, the rhonchus is usually grave, and frequently resembles the prolonged note of a violoncello, and sometimes the cooing of a dove. The natural breath-sound is more or less impaired, but that of expiration is sometimes more audible than usual, being accompanied with a wheezing. Rarely the tumefaction of the submucous tissue of some air-tubes in parts wholly prevents the passage of air; in this case the breath-sound is stopped in this part. The stroke-sound is unimpaired, and shows the vesicular structure to be free.

The sonorous and sibilant rhonchi, then, we find to be the first physical signs of pulmonary catarrh, and these are sometimes present before the cough becomes pronounced, and while the local feelings only indicate a nasal coryza, or a roughness or dryness in the throat. In slight cases, a feeling of chilliness, with sometimes tightness or pain of the chest and sore throat, are the only general symptoms at this period; but when the disease is extensive, there are rigors, followed by heat of skin, quick pulse, scanty urine, and other symptoms of incipient fever, with more or less tightness and pain across the sternum, dyspnœa, and dry cough.

After a while the inflamed membrane begins to secrete a somewhat viscid saline tasted liquid, which at first mellows the sound of the rhonchi, but, afterwards increasing, interrupts it by the formation of bubbles, which momentarily stop the vibrations, and then burst. These bubbles increase in number as the secretion increases, and are at last produced in such a continuous succession, that the sound of the former rhonchi ceases, and is replaced by a new one produced by the successive formation and rupture of bubbles in the air-tubes. This is the *mucous* or *bubbling rhonchus*. In the larger bronchi the mucous rhonchus is composed of bubbles of unequal size, causing a gurgling sound; but in the smaller tubes the bubbles are more uniformly small, and the rhonchus is more crackling; they are still, however, somewhat unequal, and accompanied by slight whistling and whiffing sounds; and, even when in the extreme bronchi, they can be distinguished to be liquid bubbles, and differ from the uniform dry crepitation that constitutes the crepitant rhonchus. The matter expectorated is now transparent and mucous, resembling raw white of egg diluted with water, and, if brought up with much coughing, contains many air-bubbles. When poured from a vessel, it falls in a stringy or ropy stream. Its viscidity is generally in proportion to the intensity of the inflammation, any exacerbation of the fever always increasing this quality. At the height of the inflammation, and at other times when the cough is violent, streaks of blood are sometimes seen; but there is never a rusty tinge, as in pneumonia.

With this state of the expectoration, the other symptoms reach their acmé. The cough and heat in the chest become troublesome, and in severe cases the dyspnœa increases to a very oppressive degree, with heaving thoracic respiration; whilst the auscultator perceives the cause in the extent of the mucous rhonchus accompanying the whole respiratory act, and the defective vesicular breath-sound. In the bronchitis of young children and of old persons, in whom the bronchial secretion is usually early and copious, the rhonchus is often universal; and this circumstance, together with a livid hue of the lips, cheeks, and nails, imports great danger, whether the sensible dyspnœa and cough be urgent or not. The chest still retains enough of air to sound pretty well on percussion, mere bronchial effusion being incapable of rendering it dull, except when conjoined with great pulmonary congestion, which takes place only in extreme cases, and is confined to the most dependent parts.*

The decline of the disease in favourable cases is marked by a change in the expectoration, which becomes more consistent and pearly, or of an opaque greenish white. This change is first perceived in the morning expectoration, and it is always accompanied by a remission of the fever, and generally of the other symptoms. The physical signs also announce the thickening of the mucus contained in the air passages, by the mucous rhonchus becoming more sluggish and clicking, from the resistance opposed to the air in passing through the inspissated liquid. This resistance increases with the increasing spissitude of the mucus, and sometimes amounts to a complete obstruction of the tube; and in this case the sound of respiration ceases in the part supplied by it. More frequently, as the mucus becomes thick, its quantity is diminished, and then it only partially obstructs the tube. This straitening of the caliber may cause a rhonchus, and being soft and incapable of vibration itself, the sound produced is a whistle, in which air is the only vibrating body. Occasionally, however, at this period of the catarrh, a ticking sound is heard, like that produced by the click-wheel of a small clock. This is caused by a pellet of thick mucus at the orifice of a bronchial ramification, which acts like a loose valve, yielding, in successive jerks, to the air pressing for passage. A change in the force of respiration may much modify these several sounds. Thus, the forcible expiration or inspiration accompanying a cough may produce the clicking sound, or even the sibilant rhonchus, in a tube which, in ordinary respiration, is totally obstructed with mucus; it may convert the clicking into sibilation, and this into the simple sound of the passage of the air; the obstacles yielding, in all these cases, to the increased force of the passing air. It is therefore useful to avail ourselves of these motions in our examination; for, by desiring the patient to cough, the nature of the obstruction may frequently be made apparent in the momentary presence of one of the above signs.

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* The anatomy of fatal cases of bronchitis, as far as it goes, is in perfect accordance with what we learn from the physical signs. The lungs do not in general collapse on opening the thorax, the escape of air being prevented by the obstructions in the bronchi. These, in most instances, contain a quantity of frothy liquid, like that expectorated during life. Sometimes it is mixed with bloody serum; but this, not having appeared in the sputa, has probably exuded from the surcharged blood-vessels during and after dissolution. Purulent matter is also observed in some very acute cases, especially those supervening in exanthematous fevers, which have proved fatal in four or five days. The mucous coat presents an appearance of an injection, diffused or in patches, of various shades, from a bright crimson to a deep brown red. It is sometimes thickened, but rarely much softened.

The uncertainty in which the signs of auscultation sometimes leave us, is completely removed by percussion. The stroke-sound of the chest is not materially impaired by catarrh; and, accordingly, the partial suspension of the breath-sound in a part of the chest in this disease cannot be mistaken for that caused by hepatization, or an effusion in the pleura.

The extent as well as the seat of the catarrh may be determined by the rhonchi. When the inflammation occupies only the large tubes, as is the case in common colds, the bubbles of the mucous rhonchus are large and uneven, and the sound of respiration may still be heard. But if the rhonchus is fine, and accompanies the whole of the respiratory act, the disease extends to the small bronchi, and, if extensive, must impede the function to a serious degree: accordingly, the dyspnœa is extreme, and the lips, cheeks, and nails, show the lividity of impending asphyxia. Cases of this kind proving fatal, are often, in this country, erroneously considered peripneumonic. In some cases of continued fever, the rhonchi indicate a bronchitis of various stages in every part of the lungs; they are the sibilant, sonorous, and mucous rhonchi; and when thus mixed, Laennec used to designate them as the *rhonchus canorus.* Their presence may be considered an unfavourable sign, and is seldom indicated by the cough or other symptoms, being, as it were, masked by the general affection of the system, which blunts all sensibility. In general, severe bronchitis is most dangerous at the extremes of life; and this depends, partly on the profuse quantity of the secretion, and partly on the inferior capability of dilatation in the pulmonary tissue at these ages; greater capacity in adults permits supplementary respiration in the healthy parts to supply the defect of the diseased. Besides these, as in all diseases obstructing the respiratory function, the dyspnœa (and hence the danger) will be great in proportion as this function is naturally or constitutionally active in the individual.

If the catarrh terminates in cure, the expectoration becomes opaque, less saline, and thicker—more *concocted*, as the ancients termed it. It is voided without irritation, in rounded, distinct pellets, consisting of an opaque, greenish, albuminous mucus. These and the cough diminish, and are confined to the morning, after waking, and a few times in the evening, and at last cease altogether. A relapse is equally marked by a return of the sputa to the glairy transparent state, which must undergo the same change before the disease is removed.

2. *Chronic Bronchitis.*—If neglected, acute mucous catarrh may assume a chronic form. The cough and expectoration then continuing, the latter is usually at first of the same quality as at the termination of the acute stage, but it sometimes becomes diffluent, less viscid, of a dirty yellow-brownish colour, and mixed with a serous fluid. The same mucus is sometimes voided in a more inspissated state, and even moulded in the shape of the bronchial ramifications,* like the

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* Cheyne, Andral, and Laennec.

albuminous exudation of the trachea in croup. This constitutes the plastic variety of bronchitis. In severe cases, the sputa frequently are mixed with pus, and sometimes become completely purulent, presenting all varieties in odour and consistence that pus, from other sources, offers. In such instances, there are sometimes quick pulse, and signs of hectic; and the disease may tend to a fatal termination with night sweats, diarrhœa, emaciation, and all the common symptoms of pulmonary consumption. These ensue most frequently on repeated attacks of acute, intense, or humid bronchitis, influenza, pertussis, and severe catarrhal affections during eruptive diseases; all of which effect a serious change in the bronchial membrane. The same change results more directly from the habitual inhalation of various kinds of dust, and occasions the severe chronic bronchitis of needle-pointers, stone-cutters, leather-dressers, and other artisans subject to this influence.*

The physical signs, when carefully studied, often afford us the only means of forming a correct opinion of these cases; but here let me caution the young auscultator against too perfect a confidence in his examinations, and too hasty a conclusion from their results. As the diagnosis is important, so is it often difficult.

The symptoms heard by the ear in chronic catarrh are the mucous rhonchus, in most of its varieties, shifting and intermitting from time to time, and occasionally the sibilant and sonorous, the presence of which is explained by the sputa; the sound of respiration, sometimes diminished, but usually unimpaired, or even puerile;† the chest, on percussion, yields a clear sound, and, examined by the eyes and hands, appears to expand pretty uniformly. It will be perceived that all these signs are negative, and none of them are characteristic of this modification of bronchitis. It is, therefore, in the absence of the signs hereafter to be described as peculiar to phthisis, that we must recognize the character of chronic bronchitis.* As, however, negative are weaker than positive proofs, so must they be multiplied to be rendered certain. If, after having repeatedly examined the patient, at different hours during several days, there are found no gurgling cavernous rhonchus, no cavernous respiration, no pectoriloquy, and no constant absence of the respiratory murmur, and of the sound on percussion, then, in spite of the general symptoms, we may, with tolerable certitude, pronounce the disease to be simply bronchial, and a still further multiplication of examinations will remove all doubt.† It is not, however, easy to get this perfect degree of negative evidence, and it requires much experience in auscultation to enable us to state it with perfect confidence. In all doubtful cases, we should take into consideration the history of the attack, the constitution of the patient, the progress of emaciation, and other symptoms of phthisical disease. The more profuse the expectoration, especially if it be purulent, or otherwise albuminous, the less likely is the case to be one of phthisis, if no physical signs of the disease are found; and it may be the more readily inferred that they proceed from a diseased membrane.

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* See Cycl. of Pract. Med. Diseases of Artisans.

† The presence of the dyspnœa, in these cases, where there is no obstacle to the entrance of air into the lungs, nay, where the puerile respiration shows it to be more perfect than usual, is ascribed by Laennec to an increased "*besoin de respirer.*" In the present instance, however, I see nothing more in this phrase than an expression of the fact. There is nothing in the state of the system that indicates the want of an increased activity in the respiratory function. The quantity and quality of the urine, and the other excrements, may be taken as pretty correct criteria of the extent of the chemical changes by respiration. I think that we must look rather to the change in the bronchial membrane, and in the nature of its mucus, for an explanation of the point in question. It is easy to conceive how a diseased state may unfit these for their office, and impair the chemical function of respiration, however perfectly the mechanical part may be performed.

CROUP.

The inflammation in bronchitis may be considered as chiefly occupying the mucous membrane of various parts of the air-tubes. In croup, the inflammation is more limited to the larynx and trachea, but it reaches deeper; the submucous cellular tissue being more engaged, the swelling and obstruction are greater at the onset of the disease, and the effusion is not mucous only, but albuminous, and sometimes in a concrete form. The earliest signs of croup, therefore, arise from the constriction of the upper part of the air-passages, causing hoarseness and a peculiar crowing cough. The constriction is at first manifest only during the fits of coughing, when it appears to be increased by temporary spasm ; but as the disease proceeds, it becomes more constant, and depends on the exudation of a tough fibrinous film into the tube. The breathing then is hurried, and accompanied, in inspiration especially, with the croupy sound ; this peculiar sound, together with a weak respiratory murmur in the chest, which yet sounds well on percussion, and, with the concave state of the intercostal spaces at each inspiration, showing that there is room for more air in the chest if it could get admittance, forms the chief physical character of unmixed croup. But it very commonly happens that there is wheezing in the chest also, from the simultaneous affection of the bronchi with either plastic or catarrhal inflammation. The sonorous inspiration of croup is audible through the stethoscope applied to the throat or upper part of the chest, before it can be heard by the ear unapplied ; we should therefore avail ourselves of this mode of examination with children, whenever they show any signs of feverishness with hoarseness. Even mild antiphlogistic measures, at this early stage, may relieve the disorder, or convert it into a common catarrhal inflammation ; but when the disease is more advanced, the most energetic treatment often fails. The paroxysms of dyspnœa, which are commonly presented even by the advanced stage of croup, must be chiefly ascribed to spasm, excited from time to time by the albuminous exudation touching the more sensitive parts of the larynx. An asthenic plastic inflammation, the *diphtheritis* of Bretoenneau, sometimes affects the air-passages in adults, causing many of the symptoms and signs of croup.

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* The mucous membrane is found on dissection to exhibit, sometimes a deep or violet red colour, diffused or in patches, sometimes little or no discoloration, and occasionally even a blanched appearance. Softening and ulceration are rare, or except in the cases arising from the inhalation of dust. Partial thickening is more common.

† This passage I have given nearly in the words of the illustrious discoverer of auscultation; yet, aware as he was of the attention required in the examination, and of the fallibility of a hasty judgment, I have more than once seen himself give proof in point by the failure of a premature diagnosis. If, then, one, from knowledge and experience so profoundly acquainted with his subject, was, through inadvertency, led into error, how much more circumspect should they be who have not his experienced tact, and his talent for improving observation?

LARYNGITIS is the croup of adults. The seat of the inflammation is the same, and it equally tends to suffocate by constriction of the passage of the larynx ; but this in laryngitis is caused by effusion wholly under the mucous membrane, none being thrown out on its surface.* The physical signs are much the same as those of croup—hoarseness, ringing or hissing inspiration, especially with the cough—whilst there is an absence of signs of disease in the chest. Although the laryngeal sound is heard also in the chest, it is easily traced to its seat by its greater intensity there; but when the laryngeal constriction has continued long enough to cause commencing asphyxia, an effusion of mucus commonly takes place in the bronchi, and may obscure the nature of the complaint, as in fact it also renders the case more complicated and hopeless. Under such circumstances, there is no wonder that bronchotomy fails to give the relief which it could afford as long as the disease was confined to the larynx.

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* This effusion is composed of lymph and serum, or pus, in the more sthenic cases. In the asthenic form of the disease it is merely serum, constituting œdema of the glottis. In cases originating from erysipelas it is sometimes a mixture of a sloughy-looking lymph with pus.

The auscultation of the breath and voice-sounds of the larynx in chronic inflammation of that organ may sometimes discover modifications of sound not perceptible to the unapplied ear; but the natural voice is generally a sufficient test of the integrity of its organ. The voice may be altered in tone, quality, or both, and very frequently it is extinguished. A deepened tone, and even a loss of voice, may indicate a more recoverable state of the vocal apparatus than a permanently raised, squeaking, or cracked voice. The former may proceed from mere relaxation; the latter implies contraction, constriction, thickening, or similar less remediable states of structure. Inability to close the glottis in coughing may be produced by ulceration and carious destruction of the arytenoid cartilages, or of their ligaments; and extensive ulceration of the epiglottis may occasion violent irritation and cough in the act of deglutition. These formidable lesions are, in the great majority of instances, the effects of syphilitic or of tuberculous disease. In the case of syphilis, the nature of the disease is to be traced in the history and in the appearance of the throat, which is generally first affected. The proofs of the existence of tubercles are to be sought in the lungs, which are most commonly their first seat.*

Bronchorrhœa, or Pituitous Catarrh.

The varieties of catarrh, which Laennec terms, from the nature of the expectoration, *pituitous* and *dry catarrh*, require to be noticed as far as they differ in their nature and physical signs from the catarrhal inflammation of bronchitis.

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* For further details see the author's treatise on Diseases of the Organs of Respiration, in the Library of Practical Medicine, vol. iii.

In the pituitous catarrh, or humoral asthma, as it is more commonly called in this country, a thin, colourless, glary liquid, is secreted in abundance by the bronchial membrane. This liquid seems to be the natural mucous secretion diluted with a considerable addition of the watery part of the blood, and some of its saline matter. This flux comes on in paroxysms, attended with dyspnœa and cough, which are relieved by a copious expectoration of the liquid, and are to be chiefly ascribed to the quantity of pituitous secretion, or to a congestive obstruction of the vessels that precedes it.

On listening to the chest at the commencement of a paroxysm, there are found a variety of whistling and droning rhonchi, which, as the attack proceeds, become cooing and chirrupping by the addition of bubbles, and at length give place to the mucous rhonchus. Very little of the natural breath-sound is heard; but the sound on percussion is not impaired except in very severe cases; and in these, the fineness and extent of the bubbling or humid crepitation show that the effusion reaches even to the smallest tubes.

This catarrh may be confined to one or two paroxysms, or it may attack daily for months and even years. Like most other serous fluxes, it is very difficult to remove when once established, and is not uncommonly dependent on organic disease of the heart, which causes obstruction to the circulation through the lungs. More rarely it arises from the developement of a number of miliary tubercles in the pulmonary tissue. Its long continuance produces that change in the mucous membrane that commonly accompanies, or is produced by, profuse watery discharges. This is a degree of atrophy, sometimes with perfect pallidity, sometimes with slight irregular striæ or patches of sanguineous injection.

These circumstances, and the fact that it most commonly attacks those of relaxed habit, languid circulation, and little disposed to inflammatory symptoms, indicate the prevailing evil to be a debility or want of tone in the vessels of the bronchial membranes, on account of which the watery parts of the blood transude with little restraint and little modification. The effects of remedies, and the nature of the predisposing causes, confirm this view. Inflammation may have been in the first instance the cause of the loss of tone in the vascular fibre; and even although it does not afterwards continue, stimulating agents may aggravate the disease, by increasing the local force of the circulation, by which a flow of liquid becomes directed to the weakened part.

Bronchial Congestion, or Dry Catarrh.

The affection to which Laennec has given the contradictory name of *dry catarrh*, is, perhaps, in its general signs, more allied to asthma than to bronchitis. The ordinary symptoms are dyspnœa, varying according to the extent of the disease, and cough, with scanty expectoration of a tough, translucent, ash-coloured mucus. The disease depends on a passive sanguineous congestion in the membrane of the bronchi, which causes tumefaction, and partial or complete obstruction in their calibre. There is with this the scanty secretion of thick mucus, which arranges itself in globules, completing the obstruction of the tube.

The stethoscopic sign of this affection is, accordingly, a suspension of the breath-sound in the part affected, whilst the chest sounds perfectly well on percussion. Sometimes the obstruction is not quite complete, and then there may be a slight sibilant or a clicking rhonchus, which can generally be made audible by the cough.

The severity of this affection depends entirely on its extent, and this may vary from a degree not at all deranging the general health, to one producing severe and oppressive asthma. Many persons, apparently in perfect health, only perhaps subject to some shortness of breath in the morning, present to the auscultator examples of the slightest degree, in a defective respiratory murmur in some part of the chest; and these usually expectorate every morning small portions of the pearly mucus that I have described. If the engorgement affect a longer extent of the bronchi, some degree of dyspnœa may be felt at any time of the day, even when the person is at rest, particularly after meals. In a severer case the dyspnœa may last for several days, and is usually relieved by cough and expectoration of a small quantity of the same viscid mucus. These symptoms are still in proportion to the extent of suppression and obstruction of respiration observed by auscultation.

This disease not unfrequently terminates in the pituitous form; or rather, its paroxysms sometimes end in a watery expectoration, with a small proportion of the tough mucus in it. Like pituitous catarrh, it may have its first origin in an inflamed state of the mucous membrane; but from the natural duration of the symptoms, and the effects of remedial measures, as well as from the appearance after death, I consider its cause rather a passive congestion, and consequently interrupted secretion, than an inflammation. It is very commonly connected with disordered digestion, or imperfect secretion of bile, with irregular alvine evacuations. There is little or no fever or pain; only sometimes a sense of tightness or heat on the chest. The tongue is often furred and œdematous; the uvula relaxed; tonsils enlarged; sometimes there are acidity and flatulence at the stomach; bowels are torpid; fæces too dark or light-coloured; the hæmorrhoidal veins often troublesome; the urine scanty, turbid, and very acid.

Excesses in diet, the sudden removal of cutaneous eruptions, suppressed gout, or sudden checks given to perspiration or any other free secretion, occasionally excite this affection. These causes, operating in a system more plethoric than inflammatory, destroy the balance of the capillary system, and produce an undue congestion or distension of certain parts of it, and impair the secreting functions of those parts. The same congestion is sometimes more directly caused by irregular action of the heart, or other obstruction to the circulation through the lungs.

Diseases affecting the motory and sensitive Functions of the Air-tubes.

Hooping-cough, or Pertussis.—This disease is both inflammatory and nervous, and in its simple form exhibits three stages; in the first it is inflammatory, like bronchitis or catarrhal inflammation; in the second it is both an inflammatory, or at least congestive and nervous affection; in the third it is entirely nervous, although it may be in some cases complicated with alterations in the pulmonary structure. It begins as an ordinary cold, early accompanied by cough, which is at first short and ringing, being apparently excited by the saline mucus secreted by the tubes and irritating the glottis. In a few days the cough becomes less frequent, but more violent and incontrollable, shaking the whole frame, and consisting of a rapid succession of violent expiratory efforts, followed by a long inspiration, which, by the hooping or crowing noise often accompanying it, may be known to be drawn through an imperfectly opened glottis. The hooping depends on an excessive irritability of the laryngeal and bronchial muscles, so that they do not relax as usual in inspiration. This sonorous back-draught is not always heard, particularly if the subject be not young: on the other hand, it often accompanies other severe kinds of cough in children, in whom the opening of the glottis is small, and disposed to contract.

On applying the ear to the chest during a fit of hooping-cough, one is surprised, with such violent external motions, to hear so little sound of respiration within the chest; and during the sonorous back-draught there is scarcely any sound of air entering the lungs. This is to be ascribed to the continued contraction of the glottis and large bronchial tubes preventing the air from entering the pulmonary texture with sufficient force to produce the ordinary respiratory murmur. In the convulsive cough of adults there is no obstructed hooping inspiration, but a full forcible one, which is heard loudly in all parts of the chest. The other physical signs of pertussis do not differ from those of mild bronchitis; there being often variable humming, whistling, and bubbling rhonchi in the upper and middle regions of the chest.

The violent and convulsive character of the cough is its most distinguishing feature. The expiratory efforts are so forcible, that the contents of the stomach are often brought up, and the face and head greatly congested. Much of the danger of hooping-cough arises from the deranged circulation in the brain, lungs, and other viscera which the fits occasion.

Spasmodic croup, *spasm of the glottis*, and *laryngismus stridulus*, or the *crowing disease* of children, all afford examples of inordinate action of the same constrictor muscles of the larynx which are concerned in causing the hooping sound of pertussis; but in these, the spasmodic contraction is independent of cough. They may be caused either by circumstances which weaken or paralyze the muscles which open the glottis, or the nerves which supply them, (the recurrents,) or by circumstances which irritate, directly or by sympathy, the constrictor muscles of the glottis. Of the former class are the causes pointed out by Dr. Hugh Ley, enlarged cervical glands, aneurism of the innominata, and thymous or other tumours under the top of the sternum. Of the latter causes of spasm of the glottis, may be mentioned a diseased state of the larynx, especially such destruction of the epiglottis as would render it ineffectual in preventing articles of food from touching the glottis: also an irritable state of the nervous system disposing to convulsions, the nicely adjusted muscular apparatus of the larynx rendering it especially obnoxious to convulsive action: this irritable state may arise from incipient inflammation of the brain, or from mere erethism connected with teething, disordered bowels, or a nervous or hysterical condition of the whole frame.

In infants, spasm of the glottis attacks suddenly, stopping the voice and breath for an instant; the child struggles, throws the head back, and gets red or purple in the face; then follows a long crowing inspiration, which shows that the spasm is becoming relaxed, and is generally succeeded by a fit of crying. There is no further physical sign; the absence of fever, the suddenness of the attacks and of their cessation, and the freedom of the breathing in the intervals, distinguish this affection from croup. The absence of cough separates it from hooping-cough. The character of the attack and the crowing sound when present, with the absence of signs of any disease in the chest, form the distinctive character of all these nervous affections.

Spasmodic Asthma.

Asthma, or *dyspnœa occurring in paroxysms*, may arise from a variety of conditions in the lungs themselves. Thus we have seen that the states of the mucous membranes constituting dry and pituitous catarrh, produce forms of asthma, especially when aggravated by intercurrent attacks of acute bronchitis. Other varieties of asthma we shall find accompany emphysema of the lungs, and extensive disease of the heart. But in nearly all these cases, the asthmatic attack is often more sudden, severe, and transitory, than the above various pathological states, simply considered, can account for; there is, in truth, a disease temporarily superadded, and this occurring separately, constitutes the complaint strictly called Spasmodic Asthma. The existence of this disease has long been *assumed* to explain the phenomena just mentioned; but until Reisseissen demonstrated the existence of circular fibres apparently muscular in the bronchi, and Laennec, by auscultation, observed phenomena explicable only on the supposition of their being affected with spasm, spasmodic asthma was merely a matter of conjecture.*

The causes exciting purely spasmodic asthma, are those which impress the nervous system, as strong or peculiar odours, irritations in the stomach or bowels, mental emotions, peculiar states of the atmosphere, and the like. The attack comes on suddenly, and, if severe, forces the patient to assume a remarkable attitude with the body bowed forwards, with the arms resting on the knees, and chest contracted, with the sensation of a weight on it, or a tight cord across it; the face becomes suffused and anxious, the veins turgid, with the other characters of ordinary dyspnœa.

During the paroxysm the chest sounds ill on percussion, and the breath-sound is indistinct, notwithstanding the violent action of the muscles of respiration. But if the patient, after holding his breath a little while, be desired to breathe again quietly, the spasm will be for an instant relaxed, and the entry of the air into the cells will be heard in a clear and sometimes puerile sound. This may be best effected in slight attacks, or where the spasm is permanent, in the manner recommended by Laennec, by desiring the patient to read aloud, or count as many numbers as he conveniently can without taking breath, and then to breathe at his ease. But after one or two inspirations, the spasm regains its hold, and the respiration becomes as dull as ever. The diminution of the respiratory noise here seems to proceed from the obstruction opposed to the entry of air into the small bronchi and vesicles, by the tonic contraction of their muscular fibres. By the same contraction the lungs are more than usual collapsed within the thoracic cavity, and the parietes of the chest being thus drawn in, pressed by the atmospheric weight on them, lose that sonorous elasticity produced by an equality of pressure on both sides. Conceiving that the contraction of the bronchial muscles would be a sufficient cause of the phenomena of spasmodic asthma, I cannot support the hypothesis of the active dilatation of the lungs, supported by Laennec and Dr. Copland. The supposed active protrusion of the lung through wounds in the chest, adduced in favour of this notion, is far better explained by the irregular pressure of the respiratory forces, in the unnatural condition of the parts, than by an assumed expansive power, of which the structure of the lungs affords not the slightest proof, and which is opposed by all we know of animal dynamics. There may be a *mechanical elasticity* in the lungs, especially in the larger tubes, which expands them after they have been unduly contracted by the antagonizing action of the bronchial muscles; but this is not vital expansibility.

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* The irritable contractility of the bronchi has been lately questioned, and is not sufficiently demonstrated by experiments. I am now engaged in some experiments for the British Association, and have already obtained results which I think will be considered conclusive on this point. See Appendix at the end.

Those who suffer much from spasmodic asthma are seldom free from a shortness of breathing in the intervals; and the frequent recurrence of the paroxysms generally brings with it more of this habitual dyspnœa. If we examine their chests, we find the same diminution of respiratory sound as during the paroxysm, but in a less marked degree; and the test of holding the breath proves that spasm exists here also, having become in a measure habitual. No doubt the frequent recurrence or long continuance of these spasmodic contractions of the tubes must lead to a permanent diminution of their caliber, and the other tissues change and fix them in this contracted size. We see the parallel of this in the irritable bladder, which, after long-continued attacks of spasm, eventually becomes permanently contracted. Where the disease is purely spasmodic, this more lasting change might not ensue for a very long period; but I have before mentioned, that with spasm of the circular fibres there is so commonly associated congestion, irritation, or inflammation, or these are so frequently induced by the spasm, that the phenomena of these pathological conditions are very commonly combined with those of spasmodic asthma. Hence, in asthmatic subjects, we may have an attack of bronchitis, of dry catarrh, or of humid catarrh, assume in its course a spasmodic character; and we may have a paroxysm of asthma, which came on suddenly as a spasm, terminate by a copious catarrhal secretion. The latter seems to be a common course of the asthmatic paroxysms, which are sometimes associated with organic diseases of the heart. The congestion which these determine in the membranes and structures of the lungs exalts their sensibility and irritability; and where the circular fibres are naturally disposed to spasm, this most readily excites it; and this spasm may not be entirely relaxed until the congestion is relieved by a free secretion from the bronchial membrane.

Paralytic Dyspnœa.

Hitherto we have considered only the spasmodic form of nervous asthma, or that dependent on an *excessive* sensibility and contractility of the bronchial tubes; but as, on examining the elements of dyspnœa, it is probable that a *defect* of these properties would disorder the process of breathing, so we are led to inquire whether there may not be a nervous asthma or dyspnœa of this kind, from weakness, or a *paralysis* of the circular fibres, or of the nerves, whose sensibility guides their contractions? We have parallel affections of the alimentary and urinary passages, when, from local or general causes, their moving fibres become torpid or paralyzed: and if I am right in supposing that the action of the circular fibres, and the elasticity of the longitudinal fibres of the bronchi, are essential to the effective performance of the act of expiration, defects of the properties of these tissues must cause a proportionate imperfection in this act. There are some facts which seem to me to bear directly on this point. Mr. Swan found, that in animals in which the par vagum had been divided in the neck, the act of expiration became imperfect, and the lungs permanently distended. Laennec remarked, that the lungs of persons who died from suffocation in sewers, appeared, on opening the bodies, uncommonly distended, and did not collapse as usual; and he proposes it as a query, whether this may not be from a sudden dilatation of the air-cells. Now, sulphuretted hydrogen gas, which is a chief component of the exhalations of sewers, is remarkable for its power to destroy muscular irritability; and it seems very probable that its direct paralyzing effect on the bronchial fibres may be the cause of the distension remarked by Laennec. We shall have occasion to return to this subject, in connexion with the lesion *dilatation of the air-cells*, termed by Laennec *emphysema of the lungs*; and we shall then advert to the view of Dr. W. Stokes, which embraces a similar pathological notion.

The phenomena of impaired contractility of the bronchial fibres would be the converse of those of spasmodic asthma; the chief difficulty being the act of expiration, which would be performed imperfectly and with much wheezing and effort, whilst inspiration might be short and comparatively unembarrassed. Now we do meet with many instances of dyspnœa, generally with old bronchitic affections, but occasionally in cases more of a generally nervous character, and in hysterical females, in which this difficulty of expiration is the prominent feature. So, also, we see the defective action of the contractile fibres of the intestinal tube arise sometimes from previous over-irritation, and sometimes from more directly weakening or paralyzing causes, and sometimes from that irregular distribution of the nervous influence that produces the phenomena commonly called hysterical. Nay, if we consider that irritations and inflammations first exalt, and afterwards injure the contractile properties of hollow organs or tubes, and that these irritations or inflammations affect successively different parts of the same tubes, we can see that spasmodic and relaxed asthma may co-exist in the same person, one part of the bronchial tubes being unduly contracted, and another unduly relaxed, from an irregular distribution of the property of irritability.

Structural Diseases of the Air-tubes.

We have now to notice certain changes of structure which the air-tubes occasionally present. These are, I believe, generally the consequences either of inflammation, or of some kindred modification of the vascular function affecting the nutritive process. Inflammation, when often recurring or long continued in the bronchial membranes, as elsewhere, induces a change of structure; and the mechanical forces to which they are subjected in the function of respiration, may modify this change in various manners.

The most simple change of structure is a mere thickening of the mucous and submucous membranes. This generally, in some degree, accompanies acute inflammation; but it is then only temporary, and subsides as the secretion becomes free and albuminous, being caused, probably, by only an infiltration of the pores of the tissues with soft lymph, which, as the inflammation subsides, is eliminated and expectorated with the mucus of the membrane. The deposits that are the most readily produced by inflammation in highly vital tissues are also the most readily removed; and thus it is, that the soft albuminous matter that is effused by acute inflammation in cellular textures, and in parenchymata in general, if it be not so abundant as to interfere with the absorbent functions of the vessels, becomes absorbed as the inflammation subsides.

But it is otherwise when the inflammation recurs frequently, or is of long duration; for it then causes an effusion of a less absorbable nature, involves the less vital structures, and, as the changes induced are slow, so they are more permanent, because they become identified with the nutritive or reparative functions of these tissues. There will then be produced a degree of hypertrophy of some or all of the various tissues composing the tubes. Sometimes there will be an extraordinary growth of the mucous membrane; more commonly, however, it is the harder and less vital textures that undergo the change, and its effect is to increase the rigidity of the tubes, so that there is a diminution of their expansibility and contractility. Nothing is more common than to see the air-tubes of persons who have long suffered from bronchitis, presenting an undue developement of the longitudinal elastic fibres; whilst in other cases the outer cellular coat of the larger bronchi is thick and indurated, and the cartilages are sometimes partially ossified. Any of these changes has the effect of rendering the lungs less easily expansible in respiration; the first in particular is a common cause of the short breath, which persons frequently affected with bronchitis generally manifest; and although not often serious in itself, yet it may so abridge the sphere of the function of respiration as to make its increased exertion, on bodily exercise, a matter of difficulty and disorder, and to render it ill able to bear any other attacks of disease, to which the lungs can in general adapt themselves by supplementary efforts.

The chief sign of HYPERTROPHY OF THE LONGITUDINAL FIBRES, and of increased rigidity of the tubes generally, is *difficulty of inspiration*, which is short, quick, and performed with an effort, especially on making any exertion; whilst the expiration is comparatively easy; but both acts are often accompanied by wheezing sounds, from irregularities in the caliber of some of the tubes, and frequently from partial congestions or inflammation, from which tubes thus diseased are rarely free. The vesicular murmur is impaired, and the expansion of the whole chest is perceptibly limited. These signs resemble those of spamodic asthma, except that they are permanent, and are not removed as the latter may be for an instant, on breathing after holding the breath, as described above.

Inasmuch as these lesions seem to arise from continued inflammation, it becomes of the more importance to direct remedies against those forms of bronchitis that are habitual, or frequently recurring. An imperfectly cured cough will often hang on a patient for months, and even for years. Not being much incommoded by it, and the general health not suffering materially, he will not think it worth while to persevere in the use of remedies, or of a proper regimen. In the process of time, however, especially under the influence of fresh colds, to which he is always more liable, the breathing becomes permanently shortened, and an irritation is often fixed in some of the affected tubes, and manifests its effect on their secreting function by an habitual expectoration, generally of a thin mucous or pituitous character. This affection varies greatly in degree. I have seen several cases of severe habitual dyspnœa, which ultimately proved fatal, present all the characters which I have described; and there was found, after death, no other lesion than a general redness of the membrane lining the larger tubes, and an extraordinary developement of the longitudinal fibres. This appearance is very common in the bodies of old people who have been long subject to cough and shortness of breath; but I have seen it also in the middle-aged, and in a few instances in younger subjects.

There is another kind of alteration of the air-tubes that has attracted more attention than those which I have hitherto described; I mean *dilatation of the bronchi.* This affection deserves attention, not only on its own account, but also because it sometimes produces physical signs which closely resemble those of phthisis. The annexed sketches exhibit different forms which the dilatations assume. To show them well, the bronchi should be laid open, from their large to their small branches, with a pair of scissors; and in examining lungs in this way, it is not at all uncommon to find the branches larger than the trunks from which they proceed, the enlargement being generally most manifest in those parts of the tubes where the cartilaginous plates are small and few; but occasionally the larger tubes are dilated also, their rings only here and there limiting the dilatation. Sometimes the dilatations are pretty uniform through some length of a tube (as in fig. 1.) In other cases they form irregular roundish cells, or pouches, freely communicating with each other, and from which tubes of unchanged size here and there arise (fig. 2.) The tissues composing the tubes are generally, at the same time, more or less altered. They are at least so in the tubular form of dilatation, (fig. 1,) in which the coats are often quite thin, and the longitudinal fibres are distinct, although occasionally enlarged. But in the more globular dilatations, the walls of the tubes are generally much altered. They are irregularly thickened ; the thickening being formed in part by hypertrophy of the mucous or submucous tissues lining the cells, which sometimes form folds or wrinkles around the tubes, and partly by a dense tissue on their outsides, probably consisting of the parenchyma of the lung compressed by the encroaching tube. There is little or no trace of the longitudinal or circular fibres in this form of dilatation, and the lining membrane is generally in a softened state, and of a red colour, whilst there may be considerable rigidity in some parts of the tubes.

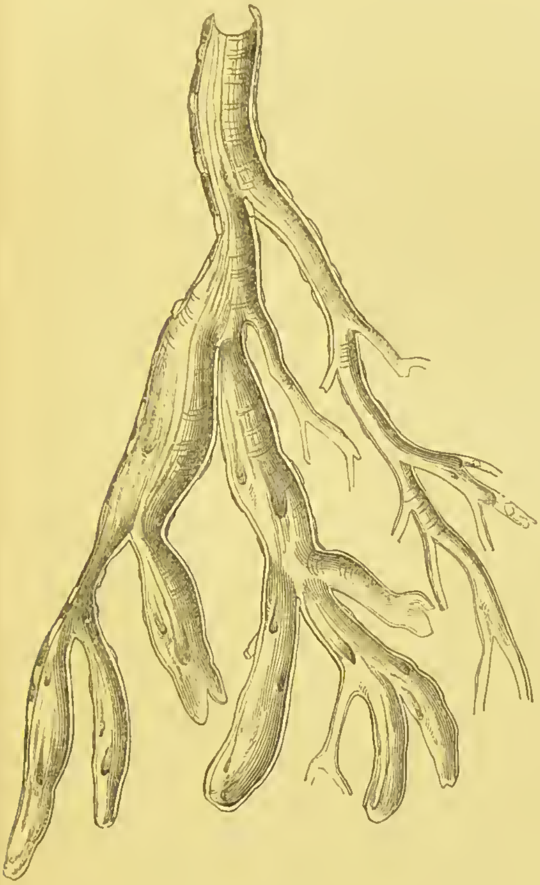

FIG. 1.

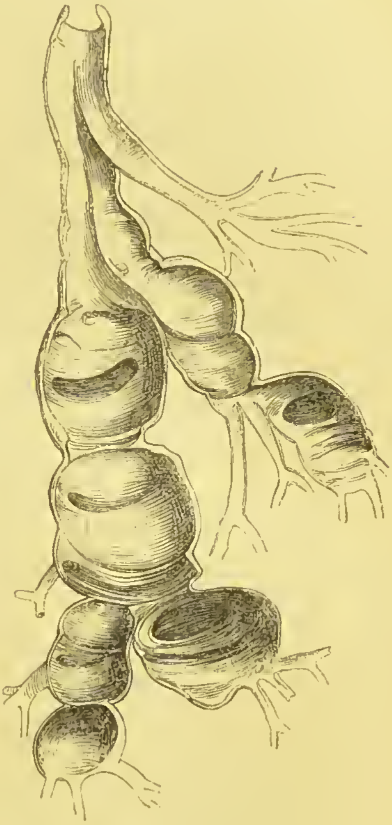

FIG. 2.

Laennec, who first described these lesions, attributed them to the frequent accumulation of mucus in the tubes, causing their mechanical distension. He considered that they were formed especially by long-continued chronic bronchitis, and that the continual recurrence of the same distension of the tubes led to their permanent dilatation. But this view is by no means sufficient to account for the remarkable changes which we frequently see in the structures of the dilated tubes; nor do these lesions constantly occur where the bronchial secretion is copious, and most calculated to cause distension. M. Andral takes a more rational view in ascribing these lesions to a modification of the nutrition of the textures composing the tubes; but he does not attempt to give any specific explanation of the mode in which their form becomes so remarkably altered.

If we bear in mind all the circumstances of the mechanism of respiration, and the various modes in which they may be deranged by the effect of disease on the textures which form parts of it, we shall find no difficulty in accounting, in several ways, for the dilatations of the air-tubes, as well as for the modifications of nutrition which accompany them, and their effects on the adjoining tissues. A mutual pressure is continually exerted between the interior of the bronchial tree and the air: in inspiration, by the air which enters to distend the tubes; in expiration, by the tubes contracting to expel the air. In forcible acts of respiration, such as coughing or energetic breathing, this pressure is increased; but in the normal condition of the tubes, when they all convey the air equally and freely to and fro, and meet the pressure with a well-proportioned degree of elasticity and contractility, this pressure is balanced and borne well; and, instead of causing disturbance, it serves to keep the air in a constant relation to the blood, and to regulate the circulation through the lungs. But disturb in any way the equality of this pressure, or derange those elastic and contractile properties which are opposed to it, and you may then convert it into a cause of unnatural distension in some parts, whilst it does not reach others with sufficient force. Now there are several circumstances that may cause these disturbances, and they are especially to be met with in those diseases which are known to lead to dilatation of the air-tubes. Bronchitis may act in both of these ways. By thickening the membranes or viscid secretions, it may cause partial or complete obstructions, which by preventing the free entry of air into some tubes, gives increased force to its pressure in others, which become distended in consequence; and it may so alter the condition of the tissues composing the tubes, that, losing their elastic and contractile properties, they yield to the pressure, and become fixed in this dilated condition. Perhaps, as Dr. W. Stokes has suggested, the mere loss of contractility may be sufficient in itself to cause dilatation of the bronchi; but I think that our view will be more satisfactory, if we take into account that this distending force is taking place in tubes softened or otherwise modified by inflammation, and that the dilatations may become perpetuated, and combined with other altered conditions of the constituent textures. Hence the lesions are often not simply dilatations of the tubes, but comprehend also irregular softenings and indurations, absence and thickenings of their several textures; so that when the lung is cut open after death, it may be at first difficult to distinguish that the irregular cavities which it presents are formed by dilated tubes. Then in the production of these dilatations we are not to forget the influence of violence in the acts of respiration. They have been observed especially to succeed to hooping-cough and other bronchial affections, in which the cough is particularly violent and long-continued. But I have met with cases of dilated bronchi in which there had been very little cough, and none of any violence; and here we must suppose that the other conditions, the irregular introduction of air and the yielding of the membranes, were more exclusively concerned in the production of the lesion. Of this kind are the following cases. In pleuro-pneumonia the lung is inflamed, and at the same time compressed by an effusion in the sac of the pleura. If it remain long in this state, the smaller air-tubes and cells become obliterated by the adhesion of their sides, so that when the liquid is removed from the pleura, they will not expand again with the enlargement of the chest: but the large and middle-sized bronchi are not obliterated; they bear the whole force of the inspired air, and become consequently dilated by it. This kind of dilatation is usually conjoined with contraction of the affected side. These cases, although not very uncommon, were first noticed by the writer.* Other lesions, which cause the obliteration or obstruction of a considerable number of the bronchial tubes and cells, must tend to produce a dilatation of the adjoining tubes, on which the motions of the chest would act with augmented force. Thus M. Reynaud observed dilatation of some tubes, when others were obliterated by the effusion and organisation of lymph. The same result has been noticed where tubercles or other tumours have compressed some tubes or portions of the vesicular texture; the adjoining tubes are dilated.

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* Lectures in Med. Gaz. 1838. Dr Corrigan has since (Dublin Journ. 1838) described cases which appear to be similar, although he has given the disease the name, *cirrhosis* of the lung.

The symptoms produced by dilatations of the bronchi will be according to the extent of the lesion. Slight degrees of it are met with in the bodies of persons who had not during life manifested any prominent disorder of the respiration; and its simpler forms may exist to a greater extent, without producing other effect than a liability to attacks of bronchitis. But where it affects many tubes, has modified their structure, and has enlarged them to such an amount that they press on and obliterate a considerable extent of the pulmonary parenchyma, it then causes habitual dyspnœa, with more or less cough and muco-purulent expectoration, which is sometimes remarkable for its fœtor. There are generally present also the ordinary symptoms of severe chronic bronchitis, from which some parts of the affected tubes are scarcely ever free; and the permanency of these symptoms, together with a degree of lividity, dropsical effusion, and cachectic condition, often induced by the crippled state of the lungs, forms the usual general character of the aggravated forms of dilated bronchi.

It is easy to understand that the air passing in bronchi dilated to a large size, or into cavities, will give a hollower, a more blowing sound, than in those of the natural dimensions: hence, over them the breath-sound may be bronchial, tracheal, or cavernous, in regions where it is naturally purely vesicular; and if, as it frequently happens, there be liquid in the tubes, the bubbling into which it is thrown will be heard to be coarse and gurgling, instead of the finer mucous bubbling of common bronchitis. So, also, the voice may be powerfully transmitted through these enlarged tubes; not in a diffused fremitus, as usual, but loud and startling, as if issuing from the spot; in some cases cracked and jarring, as in bronchophony; in others more articulate, and with a snuffling and hollow sound, as in pectoriloquy. But these are also the signs of tubercles and excavations in consumption, and we must seek for further distinctions. Besides in the history of the case and character of the constitution, these distinctions are sometimes to be found,—in the situation of these sounds, which, in phthisis, is usually in the superior parts, but in dilated bronchi in the middle regions of the chest; in their character in relation to time, those in phthisis tending to increase and spread as the excavations proceed, whilst those of dilated bronchi remain nearly stationary for weeks and months; in there being less change in the shape of the chest with dilated bronchi than with phthisis, unless they have arisen from pleuro-pneumonia, in which case the change is different; and, finally, in the nature of the sound on percussion, which, in phthisis, is more extensively dull, especially under the clavicles, whereas, in dilated bronchi, if any dulness exist, it is generally in the mammary, lateral, or scapular regions of the chest, and is often accompanied by a sound of a peculiar kind. This is a hollow tube-like sound, and from its resemblance to that produced by mediate percussion on the trachea, or by tapping with the finger on the mouth of a small phial, I have given it the name of *tracheal* or *amphoric.* We can imitate this kind of sound, by filliping on a finger pressed on the larynx or trachea, or on the cheek when the mouth is opened in the manner of sounding the letter O. This sound depends not essentially on the vibration of the walls, as in the case of the ordinary sounds of striking the chest, but on that of the air in the tubes or cavities, which give a note according to their length and size, precisely in the manner of a pan-pipe, or of an India-rubber bottle. In the natural condition of the chest we do not obtain this sound, because the stroke of percussion, and the resonance of the large tubes, are intercepted by the ill-conducting tissue of the lung: but when the bronchi are dilated so as to reach nearly to the surface, or, as we shall hereafter see, more perfectly when the large tubes are brought in contact with the walls of the chest by the pressure of a liquid effusion; or when, by perfect hepatization, the stroke and the resonance can be transmitted to and from these near the root of the lung, we then get various degrees of this amphoric sound, which it is not difficult to distinguish from the duller and deeper resonance of the healthy chest. The circumstances which favour the production of this sound are the same as those which cause morbid bronchophony and bronchial respiration, and pectoriloquy and cavernous respiration; but it requires a more perfect degree of them. Hence, although somewhat of the amphoric sound is occasionally yielded by empty cavities in phthisis, this does not often occur, it being damped by the irregularity of their form and materials, and the remains of spongy tissue on their surface. The cracked-jar sound (*bruit de pot fêlé* of Laennec) is of the same class, being a slight noise of a sudden motion of air and liquid within resonant tubes or cavities, produced by the impulse of external percussion. It is most common in large phthisical cavities, because their form and size more readily expose them to the influence of an impulse on the walls of the chest.

We shall be better able to distinguish dilated bronchi from phthisical cavities, when the signs and general symptoms of the latter shall have been described; and I shall now only add, that a case in which there have been long-continued cough with purulent expectoration, dyspnœa, loss of flesh and strength, hectic fever, even with some of the physical signs of cavities in the lungs, should not be hastily pronounced to be tubercular, if qualified by all or most of the following conditions:—If no proofs of a scrofulous habit can be traced; if the complaint have originated in a long-continued and violent cough, or in an attack of pleuro-pneumonia, and, considering its duration, emaciation have not proceeded very far; if the purulent expectoration have been fetid and sanious rather than flocculent or caseous; if the bronchial or cavernous respiration, voice, or gurgling, be heard rather in the middle than in the upper portions of the chest, and be there spread over a considerable extent of surface; if these middle portions chiefly sound differently on percussion, being dull when the rest of that side sounds pretty well, or amphoric when the side is generally dull and contracted; and if, although the cough and expectoration continue undiminished, these signs remain stationary for many weeks together.

Ulcers of the bronchi seldom occur but in connexion with some cause which concentrates inflammation in the bronchial membrane in a peculiar manner, such as the habitual inhalation of irritating particles of dust, in the occupations of needle-pointers, stone-masons, and leather-dressers; the continued passage of tubercular matter in phthisis; and occasionally the specific influence of measles, small-pox, and syphilis. I do not know of any signs by which the presence of ulcers in the bronchi can be distinguished; they rarely, if ever, exist without a similar affection of the larynx, in which case the voice is impaired or lost; but this happens commonly when the bronchi are not ulcerated, or only so far as to give vent to the matter of vomicæ in phthisis.

Tumours of various kinds are, in rare instances, developed in the windpipe, or rather in the larynx. If small, they may continue long, accompanied by violent cough and fits of stridulous breathing from the spasm which they excite in the glottis; if large or of rapid growth, they may speedily cause suffocation.

External tumours *pressing on the air-tubes* are not of very uncommon occurrence. Of those situated in the neck, bronchocele sometimes has this effect; but, as Dr. Stokes observes, it is by tumours originating within the chest, where the windpipe is surrounded by an unyielding frame of bone, that its compression is more commonly produced. Aneurisms of the aorta and innominata, various tumours of the deep-seated cervical and bronchial glands, and enlargement of the thymous gland, sometimes press on the windpipe or one of its branches, causing dyspnœa, and ultimately death by suffocation.

The signs of tumours compressing the air-tubes have been particularly described by Dr. Stokes. The usual general symptoms are dyspnœa, often in paroxyms, generally accompanied with some catarrhal inflammation of the windpipe, a feeling of constriction in the windpipe or under the sternum ; sometimes dysphagia, distension of the jugular and other veins, generally confined to one side. The chief physical signs are loss or alteration of the voice, hissing or sonorous respiration, loudest at the top or on one side of the sternum, with weak breath-sound in the lung on that side; various signs of displacement of the windpipe, the lung, and the clavicle; dulness on percussion, and sometimes projection of some of the upper portions of the chest, generally most on one side. Under the head of aneurism we shall again have to notice this subject.

CHAP. II.

DISEASES OF THE PLEURA—ACUTE PLEURISY.

Having considered what may be called the aërial or mucous surface of the lungs, and the manner in which it is modified by disease, we now come to the other surface, and the pathological properties of the serous membrane which covers it, and lines the cavity in which the lungs move. This membrane in the healthy state is thin, almost transparent, and possessed of considerable elasticity, inasmuch as it remains smooth and unwrinkled, whether the lung be expanded or collapsed. It consists of two layers: one distinctly serous, which is always bedewed with a serous fluid, lines the cavity of the chest, and forms the outer covering of its organs; the other is clearly fibrous in the costal pleura, and, together with that of the pericardium, seems to be a continuation of the deep-seated cervical fascia. An under layer of the pulmonary pleura has also been described, I believe for the first time, by Dr. Stokes; but whether this be, as he supposes, *fibrous*, or of the same structure as the outer serous layer, appears to me doubtful. It may be something intermediate between the two, for both serous and fibrous membranes consist of condensed cellular tissue, and vary only in condensation and softness, elasticity and rigidity, according to the office of the parts which they cover. The sensibility of the pleura is naturally very low; and there is neither feeling nor irritation caused by this motion of the pulmonary on the costal pleura in respiration: for a constant secretion of serum gives to both a smooth slippery surface, which reduces the friction to the least possible amount. Its blood-vessels are exceedingly fine, and probably all the larger and more visible vessels exclusively belong to the subserous cellular tissue, which are derived from both the pulmonary and the bronchial arteries.

It is chiefly these subserous vessels that form the striated patches or points of redness that are seen in the first stage of pleuritic inflammation, and their distension can be felt through the serous membrane, which feels slightly uneven on passing the finger over it. Perhaps at this period there is a diminution of the serous secretion at the inflamed spot, as we know such to be the first effect of inflammation in the mucous membranes: the effect of this would be to increase the friction between the surfaces. Very soon, however, the flow of serum is increased, and with it, if the inflammation continue, an albuminous matter, coagulable lymph, is also exuded by the serous membrane. This instance illustrates the simplest form of inflammation. We have the vessels with no compound structure to complicate or modify their action, and we find the increased developement of these vessels by inflammation obviously attended by an exaggeration of those secretive functions which they fulfil in health. These functions are twofold—that of liquid exhalation, and that of solid nutrition. The matter exhaled is serum; the material of nutrition is the albuminous or fibrinous part of the blood. In their natural proportion, these functions preserve the membrane in a healthy state; one merely lubricating its surface with a slightly albuminous fluid, the other nourishing and sustaining the solid matter of the membrane. When moderately increased by slightly augmented vascular action, these properties of the vessels cause hydrothorax and a simple hypertrophy or thickening of the subserous tissue, the most vascular part; and this result we often meet with in an opacity, without adhesions, of the pulmonary pleura or other serous membranes. But when the exhalant and nutrient properties of the vessels are exalted by acute inflammation, there is as it were an overflow of their products; the liquid effusion is rapid and copious; and the excess of the nutritive secretion now appears on the exterior of the membrane in various forms, and either by itself, or mingled with the liquid effusion, constitutes all the different products which are recognised as the result of inflammatory action. In its smallest proportion it is held in solution by the effused liquid, which, on being drawn from the body or after death, gelatinizes by this fibrinous matter which it contains. When more abundant, this forms films of lymph on the surface of the membrane; and this lymph will generally be more abundant and disposed to speedy organization, when the inflammatory orgasm is strong, and the blood is rich in nutrient matter.

Following still the pathological history of pleurisy, we find in this same lymph the matter of adhesions; but whether or not these adhesions shall take place, will depend on the quantity of liquid effusion between the pleuræ. This effusion will, of course, to a certain degree ponderate to the lowest parts of the chest, and in those parts will keep the pleuræ separate; and if it be the upper portions of the pleura that are inflamed, they will the more readily adhere, unless the liquid effusion be very abundant. But if the pleuræ be inflamed only in their lower portions, a moderate quantity of liquid will be enough to keep them separate; and if the lymph then become organized, it forms, not an adhesion, but a false membrane coating the lung, which may have further effects in modifying the remains or the products of the previous inflammation. Before we consider these various results of the modifying influence of time, of the degree and kind of inflammation, and of previous disease, on the further pathological history of pleuritic cases, we had better take a view of the symptoms and signs of the more simple and recent forms of pleurisy.

The general symptoms of pleurisy are sometimes quite characteristic: a sharp cutting pain in the side restraining every common inspiration, and often making the act of coughing or deep breathing almost intolerable; the short breath which consequently results, and the lying on or holding the affected side; short dry cough; general inflammatory fever, hard quick pulse, heat of skin, flushed cheek, &c. But extensive pleurisy and its consequences may be present without any of this array of symptoms: so that in cases in which there had been scarcely a suspicion of the presence of disease in the chest, acute inflammation and its concomitant copious effusion had been for many days or weeks occupying the pleura. And further, we may have several of the signs above described without the presence of pleurisy. Sharp pains of a nervous character not unfrequently closely imitate that of pleurisy, especially in hysterical females; and if they happen to be attended with feverish excitement, the resemblance is perfect. In fact, the greater number of the symptoms which are supposed to be distinctive of pleurisy depend on a much exalted sensibility of the pleura, which is by no means a necessary accompaniment of its inflammation; and the symptoms of oppressed breathing, proceeding from the pressure of the effusion, may be distinct only when this effusion has accumulated very rapidly.

On the other hand, the physical signs in the greater number of cases are very unequivocal; and although they by no means speak of the degree or of the intensity of the inflammation, they seldom fail to announce its presence, and they pretty accurately measure its most serious concomitant, the effusion. I will now enumerate the signs of pleurisy in the order in which they commonly occur in the affected side, and we shall then consider the nature and value of each.

1. Diminished motion and sound of respiration, from pain.
2. Sound of friction accompanying the motions of respiration.
3. Dulness on percussion in the most dependent parts of the chest, from the effusion.
4. Diminished motion, and sound of respiration, from the same cause.
5. Ægophony.
6. Cessation of vocal vibration felt by the hand.
7. Cessation of ægophony and all sound of the voice.
8. Enlargement and immobility of the side.

9. Displacement of the heart, liver, mediastinum, intercostal spaces.

10. Increased motions and sound of respiration on the sound side.

1. The respiratory movements are so far within the control of the will, that they may be restrained in parts affected with pain: and it is obvious that the sound of respiration will be diminished in proportion. This has been noticed by M. Andral as an early sign of pleurisy; but it is a very equivocal one, since it depends merely on the presence of pain, which, as I have just remarked, may exist quite independently of inflammation.

2. At an early period of pleurisy, there is sometimes heard a rubbing or creaking sound accompanying the movements of the chest. This is ascribed by Reynaud, Stokes, and others, to a defective lubrication of the pulmonary and costal pleuræ.* I am inclined to think, from a recollection of the cases in which I have heard this sound, that it always depends on the presence of lymph, and that its production is favoured by the lung being partially distended or pushed against the walls of the chest during their motions; for it is especially apt to occur where the lung is displaced by a tumour, or by a pleuritic effusion confined by adhesions, and where the lung is partially distended with tuberculous or other deposits. The friction-sound of pleurisy is commonly heard about the middle parts of the chest; it generally ceases as soon as the sound of percussion announces the accumulation of liquid: but in dry pleurisy, and in the cases of partial pressure, just alluded to, it may continue for a long time.

3. In by far the greater number of cases of pleurisy there is an effusion of serum soon after the commencement of the inflammation, and the accumulation of this liquid in the chest is the cause of the signs by which pleurisy can be best distinguished. This fluid will accumulate first in the lowest parts of the chest, floating, to a certain extent, the lung upon it. Hence these parts sound more or less dull on percussion, whilst the higher parts become unusually resonant,

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* In three cases in which this sound was heard a few days before death, I found the pleuræ thinly coated with a few small patches of soft granular lymph; and on gently rubbing the lung on the costal pleura, a perfect imitation of the friction-sound was produced by the patches, but by no other part of the pleura. The pleural inflammation and effusion was very slight, the patients dying of other diseases.

and the tone of the stroke-sound is raised. To some degree, change of posture, by changing the place of the liquid, will alter the situation of these sounds. As, however, the external vesicular structure yields more readily to pressure than the tubular parts within, the accumulating fluid soon mounts up in the form of a thin layer, between the lung and the ribs, to a considerable height in the chest. This thin layer slightly impairs the stroke-sound; and this more distinctly if the percussion be gentle and abrupt, as by filliping on a finger tightly applied, and comparing the sound, as usual, to that of corresponding parts of the opposite side. This sign, as well as those to be next described, are liable to modifications from adhesions previously existing between the pulmonary and costal pleuræ. These we shall notice afterwards.

4. This same accumulation of liquid must, as we can easily perceive, diminish the extent of the motions of respiration in proportion to its bulk, which has taken the place of the most expansible part of the lung. The breath-sound will, for the same reason, be weakened, and its duration shortened on the affected side. These signs need no further explanation.

5. About the same time at which the dulness on percussion and diminution of the breath-sound reach the middle regions of the chest, the vocal resonance there presents a remarkable modification. It is heard much more distinctly than is usual in those regions, and it is superficial, as if produced in the spot, separately from the oral voice, and changed to a sort of small bleating trembling note, which so much resembles the voice of a goat, that Laennec has well termed it *ægophony*. This modification of the voice is heard most distinctly between the third and sixth ribs, which corresponds with the situation of the middle-sized bronchial tubes; about the spine it is generally mixed with more of a common bronchophony, from the larger tubes at the root of the lung. Now the layer of liquid makes the voice more audible than usual by condensing the porous tissue of the lung, and thus making it a better conductor of sound. Then we shall understand something of the modified character of the voice, if we consider the nature of the matter which it has to pass through, a thin layer of liquid, which, being thrown into active vibrations by it, trembles and dances in an irregular manner, now checking the sound, now transmitting it with increased force, so that the voice comes through tremulous and wiry. The

high tones of the voice are those which are best transmitted in this way; for the bass tones do not enter the small tubes, but, if strong, pervade the whole tissue with a diffused fremitus. Hence we hear ægophony best in women and children, and those who have high voices. In persons with a bass voice it is more commonly limited to the inferior angle of the scapula, or near the spine, and, from its being seated in larger tubes, takes more the character of bronchophony; but even there, if the layer of liquid reach to that height, it will have something of a buzzing and bleating note. As the liquid increases, the ægophony becomes weaker, more distant, and loses much of its flutter or tremor, having now rather the sound of a diminutive, deep-seated voice, or a silvery echo of the original. This is because the lung is pushed so far away from the walls of the chest, and its tubes so much compressed; and as these circumstances increase, the sound ceases altogether. It is not easy to determine what quantity of effusion is enough to do this; but I am inclined to think that much sound of the voice is not transmitted when the layer of serum exceeds an inch in thickness. If the ægophony continue stationary for several days, it is a proof that the effusion is moderate, and does not increase rapidly, which is a favourable sign; but it is often very transient, and many cases of pleuritic effusion are discovered after they have passed the degree which causes ægophony. Old adhesions will, however, modify this as well as the other physical signs. When ægophony is most distinct, it is often coupled with bronchial respiration, especially between the scapulæ, where, also there is a good deal of common bronchophony with it. M. Reynaud has confirmed the opinion of Laennec, that ægophony is a kind of bronchial voice modified by its transmission through a layer of liquid. He observed, in a pleuritic case, that the ægophony heard at the lower angle of the scapula when the patient was sitting, became changed to simple and louder bronchophony when the patient lay prostrate or stooped much forward; this change of posture having the effect of permitting the liquid to gravitate to the anterior part of the chest, and floating the lung into contact with the parietes. Ægophony and bronchophony are different enough when their characters are well marked; but they often present mixed and doubtful varieties that do not admit of such easy distinction. As far as description goes, I would represent the true character of ægophony to be a certain *tremulousness* in the voice when it is superficial, and an echo-like slenderness when it is deep-seated; whilst bronchophony may present many other varieties.

6. An early and very characteristic effect of the accumulation of liquid in the pleural sac is its intercepting the diffused vibration of the voice, which is usually felt by the hand applied to the chest. I formerly explained that this vibration is caused by the voice pervading the common tissue of the lung, and we can readily conceive that a layer of liquid would muffle and destroy it; and it does this even when ægophony may be heard at the same spot, the vibrations of the latter being of too fine a kind to be felt by the hand. We have here also a distinction between a liquid effusion and a solidification of the lung; for the latter transmits the vocal vibrations with unusual force from the tubes. This diagnostic sign we owe to M. Reynaud; and it is the more valuable because it is so easily obtained, even by a person who does not practise auscultation. It must not, however, be always considered as quite conclusive; for there are some exceptions to it, both positive and negative. For example, in case of partial adhesions of the lung to the chest, there may be even more vibration than usual felt at the adhering parts where the lung and its tubes are pressed into close contact with the walls of the chest; and it may happen, on the other hand, in solidification of the lung, that liquid or other obstruction in the bronchial tubes may prevent the voice from being transmitted through them.

7. As the liquid effusion increases, the ægophony and all sound of the voice ceases throughout the affected side, except within two or three inches of the spine, and in spots where the lung may have been adherent, which frequently happens at the upper part of the chest. The sound of respiration is abolished in most parts of the chest, but never in the interscapular region, and rarely under the clavicle; it is, however, much weaker in these parts than on the sound side, and may probably be only transmitted from that side. I have met with several instances in which the respiration had been audible to the last under the clavicle, yet on examination after death the lung was found entirely compressed against the spine.

8. Enlargement of the affected side is the next of the signs of pleurisy that I have named. The effusion must be pretty copious to render this enlargement perceptible; but a difference between the two sides may sometimes be seen on

inspection of the chest in different periods of respiration, where the quantity of liquid is not very great. The affected side is first seen to be larger at the end of expiration, when it does not diminish equally with the other side, especially in its lower portions. So if we encircle the chest with a piece of tape, fixing it at the sternum and at the spine, we see it tighten and slacken with inspiration and expiration more obviously on the sound than on the diseased side, which remains fixed in a state of partial distension. As the effusion increases, the difference is perceptible during the whole respiratory act, and the eye can easily detect the want of symmetry, whether the inspection be made in front, behind, or, as I formerly described, from above, looking downwards on the patient's shoulders. To be more exact, however, we must measure the chest, by encircling it horizontally with a piece of tape or ribbon, making it meet at the middle of the lower end of the sternum ; then taking the tape by the point where it crosses the spinous processes of the vertebral column, we have at once the measure of two sides to compare together. Now, in comparing the measurement of the two sides, we must bear in mind that the right side is, in the healthy state, from a quarter to half an inch larger than the left; and we must not trust to slight differences in favour of the right. The enlargement of the side is sometimes discoverable by the eye, as well as by measurement, two or three days after the first attack of the pleurisy ; and it does not generally proceed afterwards in proportion to the effusion, until this becomes excessive, and has displaced the adjoining parts to a great extent: nay, I have in many instances known the enlargement diminish and disappear as the effusion displaced the softer walls of the chest; but there was still a change of shape in the chest, obvious to the eye, but not to measurement.

9. Then we have a very important class of signs arising from the displacement of certain of the walls and organs bounding the effusion. Laennec had remarked that the intercostal spaces on the side of the effusion do not present their usual depressions, and are sometimes, especially in chronic cases, even prominent beyond the surface of the ribs. This is scarcely perceptible, however, in acute pleurisy, unless the subject be thin. In such cases I have seen the intercostal spaces not only prominent, but presenting also an evident fluctuation. In looking for this sign, the patient should be placed obliquely with regard to the light; and we may some-

times perceive it more readily by surveying him from a little distance, as from the foot of his bed, than by a closer inspection.

But we may generally learn more from the displacement of the organs adjoining the effusion, especially that of the heart and of the liver. These displacements had been noticed in empyema by surgical writers; but Laennec does not seem to have paid attention to them, and I believe that we owe to Drs. Stokes and Townsend their proper application to the diagnosis of liquid effusions in the chest. The displacement of the heart by an effusion in the left pleura is the most valuable and easily recognised of these. In this case the pulsations of the heart are felt and heard most distinctly under, or to the right, of the sternum, or in the epigastrium, instead of, as usual, between the cartilages of the fourth and sixth left ribs.

Again, when the effusion is on the right side, we find the liver pushed down below the margins of the ribs, and we can trace its position both by feeling and by its dull stroke-sound. It is something depressed so much as to form a sensible tumour in the abdomen; and I have known more than one case of latent pleurisy, in which this tumour was long supposed to be the chief disease, and the case was treated accordingly. The effusion in the right side, when considerable, also displaces the heart further than usual to the left, and we may sometimes feel it pulsating to the left of the mammilla, and even below the axilla.

The displacement of the mediastinum is to be discovered only by percussion. Situated as this is naturally, in the mesial plane, it divides the two cavities of the pleura at a line down the middle of the sternum, which bone sounds well on percussion from the margins of both lungs, which lie under it. But a copious effusion will push the mediastinum towards the opposite side, and, by occupying the space behind the sternum, will give this bone a dull stroke-sound; and this may extend even half an inch beyond it. This dulness is most perceptible below the juncture of the second rib, for above this the resonance is seldom destroyed, and often has an amphoric character from the great air-tubes, which are not compressed. This is still more remarkable in some cases of adhesion, which I shall notice presently.

Now all these displacements may also be produced by an accumulation of air in the cavities of the chest; but it is plain that percussion would give a prompt distinction between these two cases, by the tympanitic sound in case of air, and the dulness in case of liquid.

10. In all cases of physical examination, we must avail ourselves of the standard of comparison which the two sides offer, and we shall often find that the sound side gives not only the negative proofs of the absence of disease in it, which may well be compared with the positive signs of disease on the opposite side, but it will even show an exaggeration of the signs of healthy action, in consequence of its work being really increased. Thus we see the healthy side move more fully and quickly than usual, and the sound of respiration will be increased there in a remarkable degree, so as to resemble the loud respiration of children; wherefore it is called *puerile.*

The diagrams annexed represent by sections of the chest the position of different quantities of effusion, as I have found them after death, and their effect on the chest and its organs. In fig. 1 there is indicated by the shading a moderate effusion on the right side, and the manner in which it displaces a lung unrestrained by adhesions; ægophony is more or less heard from *a* to *b*, and the sound of percussion is impaired, but not dull; but below *b* it is perfectly dull. Fig. 2 represents an abundant effusion on the left side, which compresses the lung against the mediastinum and spine, displaces the mediastinum and heart to the right of the sternum, gives a prominence to the intercostal spaces, and depresses the diaphragm. It might be supposed that such a quantity of liquid would render the sound of percussion quite dull on the whole of that side; but it commonly happens that percussion above *a* gives a sound which is not perfectly dull, for it receives a slight degree of resonance

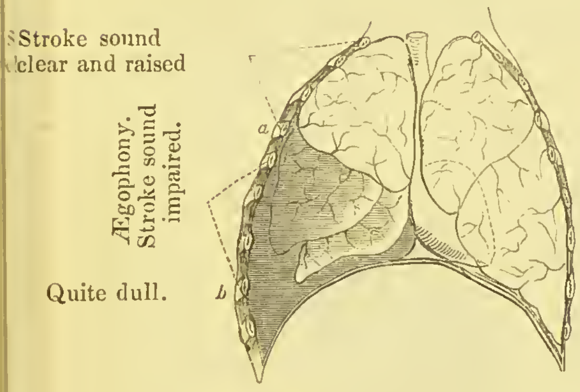


Fig. 1.

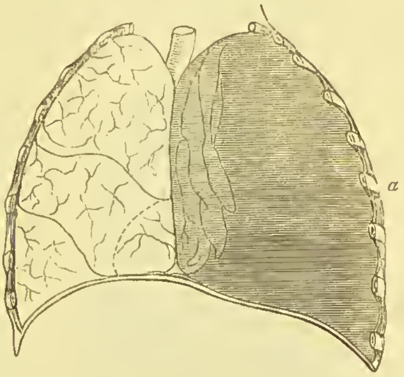
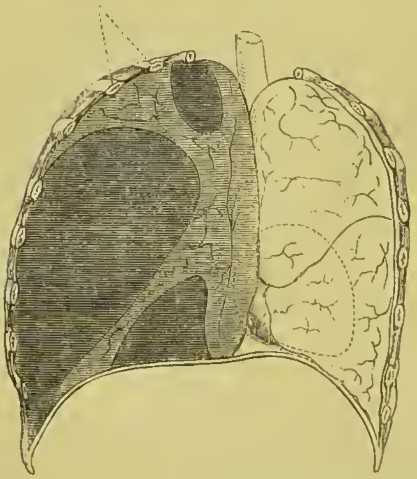


Fig. 2.

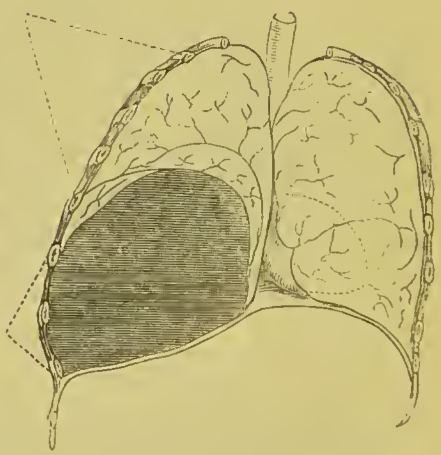
from the opposite lung; below that point the heart cuts off that source of resonance, and the sound is quite dull.

I have before stated, that the physical signs of pleurisy are liable to be much modified by old adhesions, which bind the lung to the walls of the chest; and some of these modifications are so singular and important in relation to diagnosis, that I must advert to them. When the adhesions are loose, they only form bands or cells distended with fluid; and keeping the lung at a moderate distance from the walls of the chest, they may render the continuance of ægophony much longer than it would be without them. When an adhesion is so close and strong that the accumulating fluid cannot separate it, the lung is then compressed against it; or if there are several adhering points, the attachments to these are preserved by so many pillars of compressed lung. This is represented in fig. 3, where the compressed lung still adheres to the upper part of the chest, and also to the diaphragm and mediastinum; and as the effect, instead of a total abolition of the voice and respiration, we have at the part corresponding with the upper adhesion a loud bronchophony and bronchial respiration transmitted from the large tubes by the adhering dense column of lung; and the same thing not uncommonly occurs in other of the upper and middle regions of the chest. Fig. 4 represents a pretty common form of partial effusion. The upper lobes of the lungs are the most liable to close adhesions; and when thus adhering in consequence of the former disease, the lung is compressed by the effusion below against the whole upper part of the chest. In this condition it may still admit air; but as its vesicular structure is much compressed, both the

Bronchophony. Fig. 3.



Bronchophony. Fig. 4.



breath and stroke-sounds will be tubular or bronchial, and a noisy bronchophony will be transmitted to the whole upper region of that side. I have often heard the voice and respiration quite tracheal from this cause; and I have been more than once deceived by it into the belief that there were caverns underneath. The displacement of the heart and liver, the prominence of the intercostal spaces, and the dulness on percussion of the whole lower portion of the sternum, together with the enlargement of the affected side, will generally distinguish the true nature of these cases.

A more rare form of partial pleurisy is that which affects chiefly the upper and posterior parts of the lung, the inferior being adherent to the diaphragm and lower parts of the chest. In such cases the effusion may compress the upper lobe against the front wall of the chest, and, by bringing the tubes in contact with it, occasions an uncommon developement of the tubular or amphoric stroke-sound, together with tubular breath-sound, and loud bronchophony. I have heard the stroke-sound under the clavicle in such a case, more tubular and clear than that of the trachea itself; in fact, it was quite a musical note. This has been mistaken for a tympanitic sound, and the case confounded with pneumothorax. It is often present in a minor degree in other cases of pleurisy and pneumonia.

Before we can understand the further history of pleurisy and of its consequences, we must revert to the pathological changes which accompany it.

If, in consequence of the treatment, or of the mildness of the irritation and its relief by the effusion, the inflammatory orgasm has ceased, the re-absorption of the fluid and lymph is a matter of course, and the ordinary efforts of respiration may suffice to expand the lung in proportion as this absorption proceeds. There will then be a gradual diminution and removal of the signs which marked the increase of the effusion. The return of ægophony, and of the sound of respiration, which first takes place at the parts where it was last heard, generally the upper parts of the chest, are the most available signs of improvement; and these, together with diminution of the side, and a gradual improvement of the sound on percussion, are to be watched from time to time as proofs that the improvement is progressive.

In very moderate cases, the liquid is absorbed away before the lymph or albuminous matter is removed; and when the pleural surfaces covered with this come together, a sound of rustling or rubbing is sometimes heard, which soon ends in the permanent adhesion of these surfaces by bands of false membranes. Now, if these false membranes are formed after the liquid has been removed, and the lung has recovered its full extent of expansibility, they are adapted to its free motions, and do not to any material extent interfere with them. Hence, we often find in dead bodies adhesions which are lengthened in the lower parts of the chest, where, from the action of the diaphragm, the lungs descend as the ribs rise, whilst in the upper parts the adhesions are short, because the lungs there follow more exactly the movements of the walls of the chest. But in severer cases, which are also very common, inflammation continues after the liquid has been abundantly poured out, and not only increases and perpetuates this liquid effusion, but throws out albuminous matter also in various conditions, which, by its present qualities or future changes, may produce a variety of prejudicial effects, all tending more or less to interfere with the perfect restoration of the organs to a healthy state. Now, arising, as these consequences do, out of continued inflammation, which either has been imperfectly treated, or has been neglected from being latent, they are strong proofs of the value of the physical signs, which are never absent, and which will scarcely ever fail to declare the presence of the lurking mischief. Let us now glance at some of these solid products of pleuritis.

1. Healthy and highly organizable lymph, when deposited in a thick layer, must in some measure restrain the expansion of the lung, and thereby retard the absorption of the fluid. This lymph may be diminished by absorption; and the membranes formed of it may ultimately adapt themselves to the full expansion of the lung; but there will be less chance of this, in proportion as the liquid effused is copious, and its removal slow.

2. In cases similar to that just mentioned, but with a lymph less organizable, the product of a less active inflammation, or in which there is much of the colouring matter of the blood, its organization is more tardy, and the membrane resulting is more rigid, and of a less yielding nature; consequently the lung is more permanently confined in its compressed state. The membranes which are formed on the pleura in these cases are sometimes quite cartilaginous in density, and of considerable thickness; and I have met with several examples in which they have become partially ossified. If these are formed, and acquire their density before the liquid has been removed, it is quite clear that they must for ever bind down the lung; but I have seen several cases

in which there have been proofs of contraction after the absorption of the liquid; and I should explain this by the fact, that certain newly-formed tissues have a tendency to contract for some time after their production. This is exemplified in the contraction of newly-formed skin after burns and other extensive wounds of the surface. It is also shown in the puckering of the tissue around false membranes lining cavities in the lung, in the contraction of the valves of the heart, and in many other cases where false membranes have been slowly formed, and tend to assume a fibrous or fibro-cartilaginous, rather than a serous or cellular, character.

3. But the inflamed pleura may throw out a lymph of still lower vitality, susceptible of but imperfect organization, and wholly incapable of throwing out more of an organizable character; hence, when the pleura is coated with it, if the inflammation continue, the overflow of the nutritive secretion will be in the form of a curdy matter, or of mere loose shreds of albumen.

4. The solid matter may be thrown out in a disintegrated state, utterly insusceptible of organization, and diffused through the fluid in flakes or particles, forming a mixture more or less resembling pus, which is the fluid of empyema. Although in many instances this is the result of a less active form of pleurisy, and owes its persistence or tendency to increase to the want of vitality in its solid matter, yet we do meet with cases of empyema which arise from very acute forms of inflammation. In these instances the fluid is more strictly purulent, the solid matter being in form of globules like those of pus, and seems to be the result of what may be called a suppurating diathesis, in consequence of which all the albuminous products of inflammation tend to assume a purulent character; and it is difficult to avoid the supposition that it is something in the condition of the blood that determines this less usual product from the acute inflammation of a serous membrane.

5. Lastly, as the solid accretions on the inflamed pleura we may have the various morbid productions called tuberculous, scirrhous, fungoid, and melanotic; these being commonly the result of some constitutional taint developed by the local inflammation, but sometimes also, as I believe, the product of peculiar modifications of the inflammation itself.

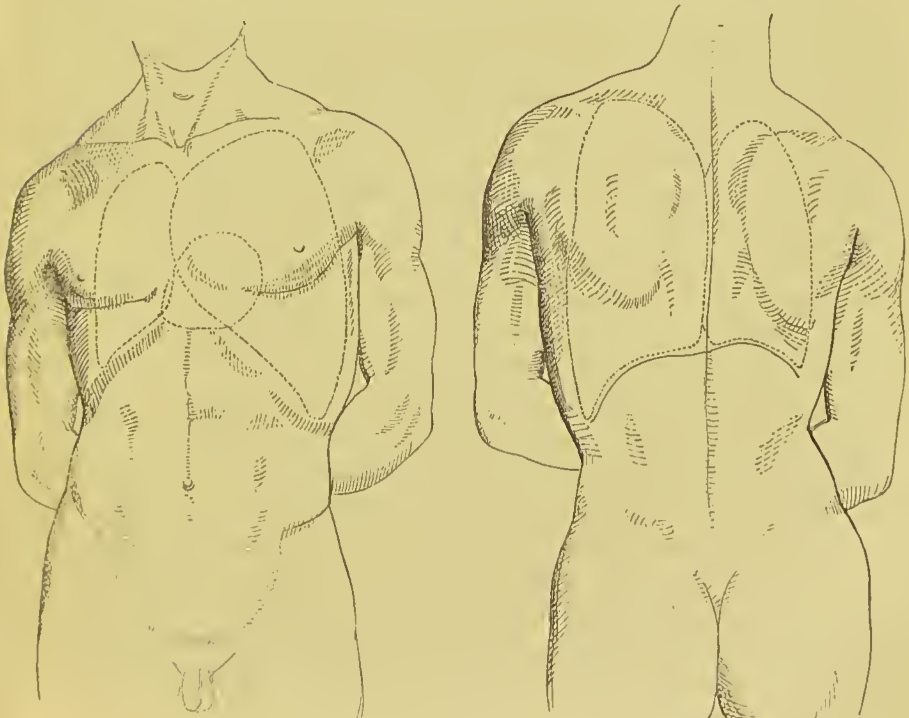
Now, it is not to be supposed that the products of pleurisy, in every case, belong exclusively to one of the kinds now described, or that they are so simple as to be rigidly divisible by such a classification. We rarely examine a protracted case of pleurisy, after death, without finding traces of several forms of the products of inflammation: we cannot examine many without perceiving that these pass by insensible gradations into one another, and that although we may meet with many that are clearly referable to one denomination, we find others in which the products of inflammation are mixed or intermediate.

If we reflect on the consequences of all these products of prolonged inflammation of the pleura, we may perceive that they all tend to keep down the lung in the compressed state to which it was reduced by the first effusion, and they do this by the rigid false membranes which are formed when the solid effusion is susceptible of organization, and by the persistence of the liquid effusion when the solid matter is destitute of organization, and acts as an extraneous irritant. We must suppose, too, that the absorbing properties of the pleura must be altered by the long continuance of disease, and that various lesions are propagated to the adjoining tissues; which, however they may have escaped the immediate effects of inflammation in its acute form, can scarcely fail to be affected by the changes of nutrition and action induced by the slow, but less limited influence of chronic inflammation. Hence the parenchyma of the lung, the bronchi, the pericardium, the bones and cartilages of the chest, occasionally become the seat of various changes. Thus the lung becomes at first consolidated and then atrophied; the tubes secrete pus and become dilated; the pericardium forms adhesions to the heart, and becomes thickened; and in cases of empyema, the ribs and vertebræ sometimes become carious and their cartilages partially ossified. Nor must we forget the unfavourable operation of the disease on other functions; the obstruction to the circulation by pressure on the pulmonary and adjoining vessels, the abridgment of the function of the lungs themselves, and the irritating or depressing influence occasionally extended from the seat of lesion to the various organs of the abdomen. In fact, besides the injury done to the respiratory organs, an imperfectly cured pleurisy may, in an insidious manner, oppress the whole system, and bring it into an anomalous cachectic state, in which morbid conditions of various kinds may be produced or brought into activity.

Now, for practical purposes, let us divide all these cases into two classes:—1st, those in which absorption ultimately predominates over effusion, and the liquid is sooner or later removed; and 2dly, those in which effusion predominates, and the liquid can only be removed through a perforation of the pleura. In the first of these, as the absorption of the fluid proceeds, what is it that supplies its place? The lung, as we have seen, is either so bound down by rigid false membranes, or so condensed and obliterated by the long-continued pressure, that it is not susceptible of its former expansion to effect this purpose. In the great majority of cases, as the liquid is absorbed away, the walls of the chest are contracted and fall in; so that the diseased side, which at the height of the effusion measured, perhaps, an inch or two more than the healthy side, now gradually becomes considerably smaller, sometimes to the extent of two or three inches. The contraction is first perceptible in the upper part of the chest, and, with the depression and more fixed condition of the shoulder, contrasts remarkably, on inspection, with the full developement and active motions of the sound side. There is still some apparent fulness of the lower part of the diseased side, and this appearance will often continue even when it measures less than the sound side. If we examine the contracted side more narrowly, we see in detail that the ribs are lower at the sides and closer together, the scapula more prominent and nearer to the spine, and sometimes the sternum, and occasionally even the spinal column also, is curved concave towards this side.

Contraction of the right side of the chest after Pleurisy.

Front View. Back View.



The dotted lines show the position of the lungs and heart.

In all this we see the results of the atmospheric pressure, together with unantagonized muscular efforts, acting on the walls of the chest. We may also have atmospheric pressure, contributing to the same end, from the abdomen: thus the diaphragm is pressed permanently upwards, carrying with it the mass of the liver on the right side, and the resonant stomach on the left; and on watching the abdomen, we do not see it swell on that side, as on the other, with the acts of inspiration. In some cases, even within the chest, the same atmospheric pressure is exerted from the sound side, causing displacements the very reverse of those which had been occasioned by the pressure of the previous effusion. Thus I have seen several cases in which the healthy lung had displaced the mediastinum towards the contracted side, not only under the whole sternum, but even under the cartilages of the ribs, to the extent of an inch beyond it; so that these parts sounded well on percussion, and the intercostal spaces there showed the movements of respiration, which scarcely affected any other part of that side. Dr. Stokes has noticed, what I have seen in several instances, that from the same cause the heart was drawn over to the right side, after a latent pleurisy affecting that side, so that it was felt pulsating to the right of the sternum.

In a side contracted after pleurisy, where the effusion has been copious and of long standing, the sounds of respiration and percussion often continue imperfect permanently, although the liquid may have been completely removed; and in almost every case they are more or less impaired for months after the attack, especially the stroke-sound; in fact, they correspond pretty well with the appearance and diminished motion of the affected side, and are to be referred to the same causes. The improvement is generally to be found first in the upper parts of the chest, and near the spine. With the return of a weak breath-sound, and slight resonance on percussion, some degree of vocal resonance may also accompany the removal of the liquid—in the superior parts of the chest amounting to bronchophony, in others being only the diffused pectoral fremitus, according to the size of the bronchial tubes, and the degree and permanency of their compression. The bronchophony in these cases is generally of a cracked or buzzing character; and I have known it very loud and diffused over some extent of surface. This is one of those instances in which some physical signs may deceive us, unless we pay attention to others, and to the general history of the case; for if, for the first time, we see a patient in this condition, and he happen to have an attack of bronchitis, we may be led to believe that the resonance of the voice and dulness on percussion are caused by consolidation, from recent inflammation in the lung, or tubercles; but this error may be avoided by attending to the history of the case, and the signs of contraction that are also present. The dulness on percussion in the contracted chest is less owing to the absence of air, than to the compressed, drawn in, condition of the walls, which are no longer free to vibrate as usual; and although there be air in considerable quantity in the lung underneath, this air gives no spring to counterbalance the atmospheric pressure, which is continually acting as a dead weight on the contracted side. Sometimes we may get more sound by pressing the fingers strongly on the side, and then striking on them; this pressure brings the walls beyond the atmospheric weight, and within the influence of the contained air, so that they may then vibrate more freely.

In these cases of pleurisy, then, the condition of the side of the chest was, at the period of copious effusion, the reverse of what it becomes when that effusion is removed; then it was dilated, and the adjoining parts pushed from it; now it is contracted, and the adjoining parts are drawn into it. Is there not, then, an intermediate stage in which neither of these conditions is presented, and the side has the usual shape and dimensions? My experience leads me to answer in the negative; for the transition from one condition to the other is not generally uniform, but partial. The common case is, that the contraction begins in the upper part of the chest, before the dilatation and displacements have ceased in the lower; and it seldom happens that there is not, during the diminution of a pleuritic effusion, an irregularity in the shape of the chest, a comparative bulging of the lower portion, which may serve to distinguish it from the case of a consolidated lung. In the cases which we have hitherto considered, the effusion was supposed to be general into the whole cavity of one side of the chest, and so was the contraction which succeeded it; but contraction cannot obliterate the cavity left by some partial effusions, limited by rigid adhesions. These cavities are in rare cases filled with air; but more frequently some of the liquid remains, and I have known it reduced to a cheesy consistence.

Now, one would think that the persons with this contracted chest, whose respiratory organs are reduced by nearly one-half, would be a crippled set of beings, capable of dragging on but an imperfect state of existence; yet it is curious enough that there have been some such individuals who have continued to enjoy good health, and to be actively engaged in the pursuits of life. Laennec tells us of the case of a distinguished surgeon of Paris, who, although he had one side contracted in a very marked degree, from an attack of pleurisy in his youth, yet enjoyed excellent health, and was in the habit of lecturing twice a day without inconvenience. I have known two or three instances of the same kind; but I must say that here the contraction was not excessive, and the respiratory murmur was by no means abolished. In other cases—and they are, I think, the most common—extensive contraction of the chest causes such an habitual shortness of breath, as to incapacitate the person from all active exertion, and to make any slight bronchitic attacks, or febrile excitement, cause severe and distressing dyspnœa. I have further had occasion to observe, that before the system becomes accommodated to the abridgment of respiration which this lesion produces, there is an enfeebled or cachectic state of the body, in which various trains of disorders may arise; and unless care be taken to counteract them by remedies and circumstances most favourable to the general health, scrofulous or dropsical disorder may be engendered, and develope new mischief in the respiratory organs, or elsewhere. Although, therefore, we may look on contraction of the chest as a mode of curative termination of pleurisy, it is one of the least favourable kind, and liable to many detracting circumstances.

We have now to advert to the other class of cases, in which effusion preponderates over absorption, and the liquid can only be removed through a perforation of the pleura. This character is to be traced, in some instances, to the condition of the membrane, which, either from its continued irritation, or from a change of structure, secretes more than it can absorb. Sometimes the accumulative tendency of the effusion may arise from some obstruction in the circulation, dependent on disease of the heart or great vessels, or even on the partial pressure of the effusion itself. But the more common cause of increasing effusion is the nature of the matter effused, which, when of a purulent character, is not readily absorbed, and constitutes the *empyema* of authors. This, when accumulating rapidly, proves a source of oppression to the breathing, and of immediate danger; if slowly, of irritation, obstruction, and consequent disorder to the vital powers generally. With the presence of pus there is not uncommonly associated an ulcerative process, which may give vent to the matter through the lungs, the walls of the chest, or the diaphragm, and which, in cases of long continuance, not unfrequently extends to the vertebræ and ribs, and their cartilages and ligaments, and to form extensive abscesses between the muscles and integuments of the chest and abdomen. The prejudicial nature of such consequences of pleurisy are too obvious to need further comment from me.

I have not given any separate notice of *chronic pleurisy;* because the transition of the acute to the chronic is so indefinite, and the symptoms of the recent disease sometimes have so little of an acute character, whilst that of a long duration occasionally manifests so much greater an intensity of irritation, that the terms acute and chronic would be less applicable to pleuritic affections than to inflammations in most other organs. And we can see some reason for this, in the fact, that the pleura being a shut sac, is liable to have its acute inflammations made a chronic disease by the retention of its product; and chronic pleurisy is perpetually liable to be excited into an acute state by the distending or irritating influence of the effusion. Still, differences of disease are very apparent in many cases, in the prevalence of high inflammatory fever in some, and in there being no fever, or one of a hectic kind, in others; in the sthenic condition of the circulation in some, and its depressed weak state in others, whatever may be the degrees of pain or nervous irritation accompanying them. These differences, although not easily included in a general view of the pathology, must not be disregarded in clinical observation, nor in the direction of the treatment.

Hydrothorax.

The serous secretion of the pleura may be increased by various circumstances affecting the circulation within the chest, such as organic diseases of the heart and great vessels, tumours at the root of the lungs, especially when these circumstances much interfere with the functions of the organs, as towards their fatal termination. But idiopathic *hydrothorax* is a rare disease; and all that has long been said about water in the chest causing orthopnœa, starting from sleep, lividity of face, &c., belongs rather to the organic disease of the heart than to the liquid effusion, which is often absent with these symptoms, and, as we have seen, is present to a great extent in latent pleurisy without producing them. Hydrothorax is sometimes produced in general dropsical states resulting from febrile action, from a cachectic state of the system, or from diseased kidneys; and when extensive, becomes, together with œdema of the lungs, the cause of oppression and danger in such cases. Slighter degrees of it very commonly take place shortly before the fatal termination of many diseases not essentially connected with the chest, and are probably the consequence of the congestion of blood caused by the gradual failure of the circulation and respiration. In all these cases the effusion may affect both sides; but it is generally more abundant on one side. So also in hydrothorax connected with organic disease in the chest, there is commonly more fluid on one side, and that not always the most diseased. I have frequently remarked that the effusion accompanying organic disease of the heart is most abundant on the right side; this is also often the case when the pericardium is adherent to the heart. The liquid of hydrothorax is simply serum ; but in other cases in which great congestion of the thoracic vessels has been its cause, we may often see in it a bloody tinge, or an approach to an inflammatory product, in a few slight loose films of lymph—proofs that the overflow extended also to the nutritive secretion of the membrane, in consequence, probably, of the temporary struggles of reaction which the vascular system frequently displays during the sinking of the vital powers. They resemble the congestive peripneumonies of the dying, which we shall soon have to notice, and should not be confounded with the products of real inflammation.

Now it is easy to perceive that the physical signs of hydrothorax are the same as those of the liquid effusion in pleurisy. There will be the dulness on percussion, and diminution of the respiratory murmur in the dependent parts of the chest, and afterwards we may have ægophony in the middle regions ; but as the effusion is seldom so extensive in hydrothorax as in pleurisy, or so much confined to one side, we do not get that abolition of the sound on percussion, and of the respiration and voice, or the displacements of organs, or the puerile respiration on the opposite side, which occur in the latter disease. The general symptoms, exhibiting a dropsical tendency, with effusions in other parts, together with the signs of disease of the heart or other organ affecting the circulation, and an absence of the general and local signs of inflammation, may generally enable us to distinguish the effusion of hydrothorax from that produced by an inflamed pleura.

Pneumothorax, or air in the pleura, may be produced in three different ways. 1. It may be the consequence of a partial pleurisy. After a pleuritic effusion has long compressed the lung, and the compression has been perpetuated by a rigid false membrane which has been formed over it, the absorption of the liquid leaves a void, which the collapse or contraction of the walls of the chest is in some few cases insufficient to obliterate, and this void is sometimes filled with air secreted by the membranes. I have seen two instances of a partial pneumothorax apparently produced in this way. They each occupied about half of the pleural sac—one the upper, the other the lower half; and the lung in both cases was strongly bound down by fibro-cartilaginous membrane, and condensed in the part contiguous to the empty space. There was also some contraction of the chest in both cases. This kind of pneumothorax is very rare, and I do not think that it is described by any author.

2. Another kind of pneumothorax is that which may be called idiopathic, and arises from an effusion or secretion of air into the sac of the pleura without perforation. This is also of very rare occurence. It is said to be sometimes met with towards the termination of fatal diseases, in the same manner as tympanitis occasionally occupies the peritoneal sac under similar circumstances. I cannot say that I have ever met with a case in which I discovered any signs of such a form of pneumothorax during life; although I have several times seen air in the pleural sac on opening bodies after death, where no perforation could be discovered in the pleura. It is possible that the air may have been exhaled from the animal fluids after death, and then increased by exosmosis through the lung. The facility with which air pervades dead membranes countenances such a notion. Pneumothorax is also said, by Dr. Graves and others, to have occurred in a few instances at the commencement of pneumonia, and to have soon afterwards disappeared; but as the chief sign in these cases was a remarkable resonance on percussion, I suspect that these were examples of the production of tracheal or amphoric sound on percussion, from consolidation of the upper lobe of the lungs, and not cases of pneumothorax.

3. By far the most common kind of pneumothorax is that caused by some unnatural communication between the pleural sac and the external air; and this may be by a perforation either of the external parietes or of the pulmonary pleura. The latter case is the now usually recognised

cause of pneumothorax, and constitutes the great bulk of the examples that are met with. The perforation depends on the progress of ulceration, generally of a tuberculous character, rarely of gangrenous abscess, through the pleura. The circumstance of ulceration reaching and perforating the pleura, indicates a low state of the reparative powers, and a want of plasticity in the products of inflammation ; for, under ordinary circumstances, ulceration could not approach the pleura without causing it to inflame and throw out coagulable lymph, which, becoming organized, forms either a protecting thickness of membrane, or close adhesions to the costal pleura. We see this in most cases of chronic phthisis, where the upper lobes are generally adherent to the ribs. I have seen an ulceration extend from a tuberculous cavern across the two layers of the pleura, thickened and adherent, and completely through the walls of the chest ; so that when the patient coughed, air bubbled out of two or three fistulous openings in the front of the chest; but here was no pneumothorax. On the other hand, I have met with more than one case in which the adhesive process seemed quite incapable of protecting the pleura, which was consequently perforated in several points ; wherever, in fact, the ulceration of the lung extended to it, and the air freely entered the sac of the pleura by all these holes. More commonly, however, there is only one perforation, and this is generally near the apex of the lung in connexion with some of the cavities that first form there.

The completion of the perforation is in most instances sudden ; a part thinned by ulceration, and imperfectly adherent, giving way during a fit of coughing, or some other unusually forcible act of respiration. I have known it caused by external violence. The immediate effect is to admit air more or less rapidly into the pleural sac, which, by equalising the atmospheric pressure outside and inside the lung, permits it to assume that state of collapse to which its natural elastic contractility would reduce it. Hence dyspnœa, sudden and severe, in proportion to the extent to which the air enters and the lung becomes collapsed. But the access of atmospheric air to a serous membrane totally unaccustomed to it, also occasions great irritation and consequent inflammation. Hence a sudden sharp pain and dry cough, with spasms of the intercostal muscles, and a weak, quick, and sometimes irregular pulse. Soon the irritation becomes accompanied with inflammation, and then follow the symptoms of acute pleurisy, with heat of skin, and inflammatory pulse; and a liquid effusion is added to the air in the pleural sac.

Now, although a perforation of the pleura will not fail to introduce air into the pleura, the amount and effect of the introduction of this air will vary considerably, according to the size and other circumstances of the ulcerated aperture. If this be very small, or if, as it not unfrequently happens, it be so placed that the walls of the chest close it in expiration, by which it is rendered valvular; or if it be below the level of the liquid, the air introduced by each inspiration will not escape as freely in expiration, and the result will be progressive accumulation of air in the pleura, and a consequent increase of compression of the lung and of the dyspnœa; and in this way perforation of the lung has, in some cases, caused suffocation within a few hours of its occurrence; in others, this catastrophe has been delayed by the vent given to the air by accidental changes of position, or the effect of violent cough, or by puncturing the chest. If the aperture be of larger size, and no impediment occur to the passage of air through it, it will interfere with respiration only so far as it suffers air to pass outside of, instead into, the lung. But when the air passes thus freely, the pleura is more irritated by it, and there is a more copious secretion of liquid, which is generally purulent, and often fetid.

The physical signs of pneumothorax are generally very remarkable and distinctive. The presence of air in the pleura will give to the walls of the chest a freedom of vibration, and therefore a degree of resonance on percussion, even greater than that which the air-filled structure of the lung confers on them; so that percussion will give more of the drum-like note or tone which we get by striking over the region of the stomach or of the cæcum. This will be more marked in proportion as the quantity of air is considerable. The same circumstance will also impair or destroy the sound of respiration; for the air not only removes to a distance the pulmonary structure where this sound is produced, but also by its pressure diminishes that entry of air into the cells on which the sound depends. There will be, therefore, this remarkable contrast of signs to distinguish pneumothorax—a good or hollow sound on percussion, with little or no sound of respiration, whilst the opposite side gives a worse sound on percussion, but a much more distinct respiratory murmur.

But there is another class of sounds produced in air-filled cavities, which may often give decisive evidence of their existence. If we apply the mouth of an India-rubber bottle to our ear, and observe the sound which striking on its outside causes, we shall find that each stroke is accompanied by a little tinkling note, like the clink of a piece of metal or glass. This note is an echo produced by the reverberations or repeated reflections of the impulse from the walls of the cavity, and it is shrill and acute, because the reflections are short and quick, in so confined a space. It is the same sort of note that we hear on listening to the mouth of an empty cask, but it is there less shrill, because the space is larger. Any sound proceeding from, or communicated to, the cask, the India-rubber bottle, or any cavity in the body with reflecting walls, will be accompanied or followed by this sort of tinkling or ringing echo, which will be more prolonged and distinct in proportion as the walls are perfectly and uniformly reflecting. We often hear sounds of this kind, on using the stethoscope over the stomach or large intestines, as their contents move and cause a sound within them. So, too, we find the tinkling echo accompanying the sounds proceeding from an air-filled cavity in the chest, and it becomes a distinctive sign of the existence of such a cavity.

In idiopathic pneumothorax, and in that partial kind resulting from the absorption of a partial pleuritic effusion, although the cavity be present, there may be no sound produced in it, or transmitted to it, so as to cause the tinkling echo. Sometimes percussion on the external walls will do this; but care must be taken not to strike too near the spot where the stethoscope is applied, or the clink sound produced by abrupt impulses on the tympanum may be mistaken for the tinkling echo. Sometimes the voice or cough may be transmitted to the cavity by the condensed tissue of the lung. I have heard the metallic tinkling accompany both of these in a case of partial pneumothorax, in which there was neither liquid effusion nor perforation of the pleura. But it is where liquid is present, and where the pleura is perforated, that we most commonly hear the phenomenon of metallic tinkling; not, as Laennec supposed, because these are essential to its production, but because the motions of the liquid, or of the air through the orifice, make sounds within the cavity, which serve to show its echoing properties. So metallic tinkling has often been heard after the operation for empyema, showing the presence of air in the pleura.

Perforation in the pleura, with its consequence, pneumothorax and liquid effusion, is not a very uncommon accident in the course of phthisis; and its physical signs are so remarkable that they can scarcely fail to be recognised even by those who are but moderately versed in auscultation. The tinkling reverberation may present several modifications, which it is useful to notice, as they may serve to give a more accurate knowledge of the condition of the parts, and of their tendencies. When the perforation is small, or obstructed by its position against the walls of the chest, or below the level of the liquid, the tinkling is seldom heard except on coughing or taking a full inspiration, which reaches the cavity, and may throw the liquid into bubbles. The voice may also sometimes find access to the cavity through consolidated portions of the lung, and then it will be accompanied by a tinkling. When the orifice is large and free, the air will pass in and out in ordinary breathing, and will produce in its vicinity a sound like that of blowing into the mouth of a glass bottle; and this kind of respiration is therefore called *amphoric*. In such cases there is seldom so much oppression of the breathing as in those where the air passes less freely, and accumulates in the cavity. In listening for the tinkling phenomena, it must be recollected that they may be audible only in certain parts of the chest where the lung is not adherent, and where the liquid effusion does not reach. Generally, in the sitting posture, they are heard best about the mamma, and in the region of the scapula; but I have heard them in every part of the affected side, and sometimes only in one spot. In fact, there must be a certain degree of tension in the walls of the cavity to make them good, reflecting surfaces; and if this be deficient at the spot opposite to that on which the stethoscope is applied, the sound may be absorbed, and not reflected.

The addition of the liquid to the air in the chest makes the diagnosis still more easy. Percussion indicates the exact level which separates the two, and we find that change of position varies the position of the liquid much more distinctly than when there is no air in the pleura. The motions of the liquid also may give very decisive evidence of its presence in a free cavity. On change of posture, or on coughing, the liquid will sometimes drop from the parts which have just been immersed, and the sound of this will exhibit the metallic ringing in so distinct a manner, that it resembles the note which a glass or porcelain vessel gives when struck. If the liquid be agitated more forcibly, as by the patient giving his body an abrupt jerking half turn, it can be heard to splash most distinctly against the walls of the chest; and this is the sign of *succussion* which was described by Hippocrates. This may also be accompanied and followed by the tinkling echo. The splashing is not easily heard, unless there be a good deal of air in the pleura, and a certain quantity of liquid. The proportions of these are, however, better to be ascertained by percussion. Dr. Ogier Ward has remarked that the consistence of the liquid may sometimes be ascertained by the facility with which the splashing sound may be produced; this diminishing as the liquid becomes thicker and more sluggish.

CHAPTER III.

DISEASES AFFECTING THE TISSUE OF THE LUNGS.

Pneumonia, or Peripneumony.

Peripneumony consists in an inflammation of the parenchyma of the lungs, and, according to the changes produced in the tissue, it is divided into three stages.

The first is that of simple inflammatory injection, in which the size and apparent number of the blood-vessels is greatly increased. Our knowledge of minute anatomy does not permit us to specify with certainty the exact and essential seat of this inflammation; but I am disposed, from a consideration of the signs, and from many minute examinations of the effects on the tissue, to refer it principally to the important plexus of the blood-vessels, which in fact constitutes a chief part of the parenchyma of the lungs. It is its seat in this plexus which, as constituting the organ of the minor circulation, through which the whole blood of the body must pass for a vital purpose, stands in most peculiar and important relations to the functions of the heart between whose compartments it is the communicating channel—that gives to pneumonia its remarkable and serious character. From this plexus it usually extends more or less to the mucous lining of the bronchi, and to the pleura investing the inflamed part; but these extensions are uncertain and trivial compared with the essential disease of the vascular parenchyma.*

In this stage of the inflammation, the distended vessels, and perhaps a serous effusion in the interstices, press on the minutest bronchial ramifications, and partially obstruct the ingress of air into the cells to which they lead; whilst the viscid secretion of the mucous membrane simultaneously inflamed, yields to the air in respiration forcing its way through it in successive bubbles. At first the obstruction may only give a slight roughness and sharpness to the breath-sound; but as it increases, the bubbling is continuous. This bubbling passage of air through a viscid liquid, contained in an infinity of tubes of equally diminished caliber, causes that fine regular crepitation which constitutes the true *crepitant rhonchus.* If the inflammatory injection be not so general as to prevent the air from entering without obstacle into many of the bronchial cells, then, besides the crepitation, the natural breath-sound will be heard. On the other hand, the inflammatory engorgement increasing, causes a total obstruction of the cells, and all sound of respiration with crepitant rhonchus is confined to the end of each inspiration, or to the act of cough, and at length ceases. The progress of the inflammation is, therefore, now marked by the gradual cessation of the crepitation.

The sound on percussion is only slightly impaired in the first part of this stage; but as the engorgement advances, and the proportion of air in the inflamed spot of lung is diminished, the sound becomes more dull in the corresponding part of the chest. If the disease be extensive, manual examination may also often detect a deficiency in the motion of the ribs on the affected side.

The general symptoms of this stage are, fever, with more or less pain in some part of the chest, cough with viscid expectoration, accelerated and sometimes oppressed breathing. The decubitus is generally on the back; the pulse quickened, but very variable in its character; the fever is generally of the inflammatory kind, but is occasionally typhoid.

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* On dissection, the lung in this stage is found to be of a deep red colour, of various shades; it is increased in weight, and pits on pressure, but it is still somewhat crepitant, and usually floats in water. When cut into, it still presents its spongy structure, out of which generally exudes abundantly a spumous bloody serum. Sometimes it is softer and very livid, and only a little spumous blood flows from its cut surface. Its integral cohesion is diminished, for the texture may be easily broken down between the fingers. The mucous membrane of the small bronchi is of a deep-red colour.

Pneumonia modifies the secretion of the bronchial mucous membrane in a very remarkable manner. At the commencement of the disease there is frequently no expectoration, or it is simply catarrhal, being composed of a mucus of moderate tenacity; but as the crepitant rhonchus becomes marked, the sputa assume their characteristic form. They are semi-transparent, tenacious, and run together, forming one mass of a reddish yellow, or rusty tinge of various shades. As the disease advances, this tenacity increases. At first it does not much exceed that of the white of an egg, and when poured out, the sputa fall in glutinous strings, but at the height of the first stage they are frequently so viscid, that inverting the vessel, and even shaking it in this position, will not suffice to detach them from it. The same tenacious property imprisons in the mass a multitude of little air bubbles, which sometimes produce a spumous appearance. The colour may vary, in numberless gradations, from a light reddish or greenish yellow to a deep orange, a dingy red, or rusty hue. All these tints proceed from various proportions of the colouring matter of the blood, which, transuding from the engorged pulmonary vessels, becomes intimately combined with the secretion of the bronchial membrane. Quite different from these are the sanguinolent sputa, that sometimes occur in catarrh, in which the blood appears in distinct striæ. The intimately combined tint, and the glutinous viscidity of peripneumonic expectoration, give to it a character which is generally distinctive, and sufficient, with the febrile and other symptoms, to prove the presence of the disease. Moreover, the degree of viscidity in some degree announces the intensity of the inflammation; and whenever, after having become thinner and partially opaque, in the course of cure, the sputa regain their former viscidity, and semi-transparency, a relapse of the disease is indicated. But although the presence of these sputa indicates with certainty the existence of pulmonic inflammation, we cannot draw an opposite conclusion from their absence. They rarely appear until the second or third day, sometimes not till later, and in some cases have not been observed at all.

The second stage of peripneumony is that in which the lungs present that change in the tissue which is called by Laennec hepatization.* This change consists in the gradual effusion of a semi-solid albumen in the interstitial tissues, which, pressing on and obliterating the cavities of air-cells and smaller bronchi, destroys the spongy texture of the lung, and

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* *Remollissement rouge* of Andral.—A hepatized lung presents the following characters after death: Externally it is of a deep-red colour, which internally is studded with a number of lighter small granular spots, with patches of whiter colour, marking the vessels, interlobular septa, &c. less affected with the inflammation. It sinks in water, and is no longer crepitant but inelastic to the touch, breaks readily under the fingers, and may, by a slight pressure, be reduced to a red pulp. When cut into, it is often mottled, and paler than in the first stage. The little granular points are best seen on tearing the lung, and they then seem to be the most solid parts. They appear to consist of the little bunches of vesicles, (in which, according to Reisscissen, each minute bronchus terminates,) whose membranous tunics have become so swelled by the deposition of a soft albuminous matter in them, as well as from the increased size of their blood-vessels, that their cavities are obliterated. For a full consideration of this and other parts of the anatomical and pathological history of Pneumonia, the reader is referred to the author's article on that subject in the Cycl. of Pract. Med., from which I will insert here one extract.

"On inspecting, by aid of a simple lens, the margin of a slightly inflamed spot of lung, numerous vessels may be seen, distended with blood, passing across, around, and between the vesicles; and as the scrutiny is extended to a part more inflamed, these vessels are so multiplied and confounded with each other, as to be no longer separately discernible. In this state, it is impossible to distinguish whether the tunics of the cells, or the tissues which connect them, are most affected, for they all appear one mass of redness, in which are seen the cells irregularly diminished in size, and containing bloody serum with bubbles of air. The interstitial cellular texture, where it can be separately discerned, namely, between the lobules, and around the larger bronchi, is generally less vascular, and of a lighter colour than the other parts, and in some instances is nearly free from the inflammation. The lining membrane of the minute bronchi, although generally of a deep-red colour, is sometimes bluish-red, as if from redness under it, rather than in it; and, on tracing these tubes higher, the presence of the inflammatory mark is very uncertain. These examinations, and some pathological considerations, induce me to regard the capillary ramifications of the pulmonary artery and veins as the proper seat of pneumonia, and that these may involve more or less of the tissues through and around which they pass. Thus, through them, the tunics of the air-cells, particularly the submucous, commonly become the seat of inflammation, whence are formed the granulations of ordinary hepatization. When, again, the inflammation is confined more to the intervesicular plexus and tissue, which appears to be the case in the more congestive form of inflammation, where vessels larger than capillaries are involved, a uniform, non-granular form of hepatization is produced. If, as is commonly the case, the inflammation has extended to the extremities of the bronchial arteries, which are said by anatomists to anastomose with the pulmonary, the mucous membrane lining the vesicles, and minute bronchi, partake of the inflammatory action, and exhibit it, in the manner peculiar to mucous membranes, by the secretion of a viscid mucus, and afterwards of pus. So likewise, when the inflammation reaches the surface of the lung, it is generally, but not constantly, propagated to that portion of the pleura which invests it, and derives its vessels from it, and this extension of the inflammation may give a pleuritic character to such instances of the disease. But it is in the extensive capillaries of the lungs, through which the blood of the whole system is continually passing,—it is this affection of so considerable and important a portion of the circulatory system in the immediate vicinity of the heart, that causes the severe and intense character of pneumonic inflammation; and the more constantly we hold this in view, the better shall we understand the pathological history of the disease, and its important relations to remedial measures."

In connexion with these views of the minute pathology of pneumonia, may be considered the physical cause of the crepitant rhonchus, on which various opinions have been formed; and in doing this, as some subjects must be anticipated, I would recommend the student not to read this note, until he shall have duly studied the subsequent sections.

It is agreed on by all auscultators, that œdema of the lungs, and the margins of hæmoptysical engorgements, may produce a rhonchus of the crepitant kind; and it naturally occurs to us, that these lesions resemble pneumonic engorgement, in the pressure to which they subject the vesicular parenchyma. If we compare with these instances the very close representation of the crepitant rhonchus, which the simple pressure of a healthy lung will produce, we are led at once to an explanation of the mechanism of this sign. This pressure forces the air from the tissue, but it so narrows the tubes and cells, that the air can only pass out of them in successive bubbles, the escape of which produces minute crepitations. This sound, Laennec tells us, differs from the crepitant rhonchus only in not being so strong, there being no preternatural fluid present in the air-cells. In œdema, or in effusion of blood into the substance of the lungs, we have interstitial effusion and narrowing of the minute air-tubes and cells, and from the nature of the expectoration we may conclude that there is also an increase of liquid within them; the rhonchus that accompanies them is accordingly described by Laennec to consist of moister and somewhat larger bubbles, and he terms it subcrepitous. Lastly, we have the peripneumonic engorgement, in which anatomy assures us that there is the same narrowing of the air-tubes and cells by the swelling of the interstitial blood-vessels, whilst the observation of the sputa leads us to conclude that the interior of these tubes is during life lined with a viscid secretion;—this produces the drier and stronger sound of the genuine pneumonic crepitation. I have given the description of the sounds on the authority of Laennec, because, as a practical auscultator, he has certainly never been surpassed; and yet he had no *fixed* views as to the cause of the crepitant rhonchus. (See Dr. Forbes' Transl. pp. 52, 212.) M. Andral considers this sound to be nothing more than a finer modification of the mucous rhoncus, the latter becoming crepitant when its seat is in the minutest bronchi and vesicles, the narrow dimensions of which render the bubbles which compose it finer and more equal. This has also been ingeniously advocated by M. Piorry, and in this country by Dr. Spittal and others, who consider the crepitation to arise from the bursting of minute bubbles in a serous liquid, the same probably as that which flows after death from an inflamed lung, when an incision is made into it. I have doubted the accuracy of this explanation, because the character of the expectoration in pneumonia does not warrant the supposition that there is during life any other secretion into the air-cells in the first stage than the characteristic viscid secretion of the mucous membrane which lines them. I have before pointed out the tendency which the natural respiratory movements have to throw all superfluous secretion from the smaller bronchi into the larger, until they are brought under the influence of the act of expectoration. Now if, as those who found their opinions solely on morbid anatomy maintain, there were a serous effusion into the air-cells in the first stage of pneumonia, there ought to be more or less of this serum mixed with the sputa, as in some cases of pulmonary œdema; this, however, is not the case, for the expectoration is a glutinous mucus from the onset of the inflammation, and, instead of becoming more serous, increases in viscidity as the inflammation becomes more intense. Moreover, we venture to assert (after Laennec) that there is a mucous or liquid rhonchus of the fine bronchial tubes, the character of which is sufficiently distinct from the rhonchus of pneumonia to merit its separation in kind as well as in degree from the latter sign. This fine mucous rhonchus, which I have observed in pituitous catarrh, and in the converts it into a more or less solid mass. Such a condition of the air-cells precluding any further ingress of air into them, what stethoscopic signs can we have to indicate this stage of inflammation in the living body? Here still a consideration of the physical state of the organ will teach us to expect, *à priori*, the same phenomena that experience has revealed. We have already had occasion to observe that the healthy lung, from its being composed of conductors of very different powers, (air, membrane, and liquid,) is a bad conductor of sound, and is, therefore, incapable of transmitting to its surface slight sounds, remote in the interior. But now that the tissue is rendered more uniformly dense by hepatization, it becomes a better conductor, and transmits a sound (usually unheard) of the air passing to and fro in the larger bronchial ramifications. This is the *bronchial or tubular respira-*

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general bronchitis accompanying continued fever, occupying the base of the lungs, the common seat of the crepitant rhonchus, is distinguished from this by the greater inequality of the bubbles which compose it; they appear to roll through a liquid, without breaking with that regularity which distinguishes the crepitation in pneumonia. There are, besides, little hissing or whistling sounds mixed with them, which convey to the mind the impression of a moveable proportion of air and liquid in the tubes. If this submucous rhonchus is to be distinguished from the subcrepitant of pulmonary œdema and apoplexy, it is by the greater irregularity of the minute sounds which constitute it; but there may be gradations of the two which cannot be distinguished. But I still maintain that the crepitant rhonchus of peripneumony is, with few exceptions, sufficiently characterized by its pure equal crepitation, unmixed with hissing or any sounds of liquid, to render it a valuable and available means of distinguishing this disease in its earlier stages. In conceiving the mechanism of this rhonchus according to my explanation, we must take into account the force with which the air passes through the narrowed tubes, and we shall then perceive why the bubbles crepitate drily, and the liquid is not carried before the air passing to and fro, as it would were its viscidity less, and its quantity greater. It is stated by Dr. Spittal of Edinburgh, that the crepitation of peripneumony may be most closely imitated by the bursting of bubbles on the surface of fluids of the tenacity of serum, and hence he ingeniously deduces that the crepitant rhonchus depends on the mixture of air with such a fluid in the lungs. But I must remark that the cases are not analogous: in the one, the bubbles rise and burst merely from their own levity; in the other, an active moving force is constantly driving and breaking them through an infinity of minute tubes. As there is no serous expectoration in the first stage of pneumonia, I cannot but regard the serum found in the lung after death, as in great measure the result of the cadaveric separation of the serum from the blood in the engorged vessels; and I would still, as formerly, refer the crepitant rhonchus to the forcible passage of air through the narrowed air-tubes and cells lined with a viscid secretion. As the vesicular parenchyma becomes obstructed by the deposition of albuminous matter from the inflamed vessels, this crepitating entrance of the air becomes less, is heard only at the acmé of inspiration, or during the more forcible efforts of coughing, and at length, when the stage of hepatization is complete, ceases altogether.

I feel great satisfaction in adding, that Dr. Chambers has long entertained opinions on the crepitant rhonchus of peripneumony very similar to mine, and he does not admit that a serous effusion accompanies the commencement of the inflammation.

tion of Laennec and Andral; and marks the advance to the second stage of pneumonic inflammation. This sound, when once heard, cannot be mistaken. It resembles that produced by blowing through a crow's quill, and is frequently so loud as almost to amount to a whistle; acute and defined, it forms a remarkable contrast with the dull, diffused sigh of vesicular respiration. In other parts of the lung, if the disease is extensive, the breath-sound is puerile.

Another nearly as characteristic sign is given by the voice. When the stethoscope, with its stopper in, is applied to the diseased part, the voice is heard to resound there in a tone modified, as if speaking through small tubes. The voice does not, as in pectoriloquy, appear to enter the tube of the instrument; and the sound of the voice is not heard in distinct words, but in notes of various continuance, not always synchronous with the words uttered by the mouth; and the intervals are often alternated with what may be called whiffs of bronchial respiration. It is obvious that the extent and intensity of these sounds must greatly depend on the number and size of the bronchial tubes in which they are heard. They are therefore most distinct when the hepatization occupies the summit or the neighbourhood of the root of the lung, and extends to the surface. On the other hand, when the surface or the centre alone is hepatized, these signs may be altogether wanting.

The sound on percussion is now, and in the subsequent stage, perfectly dull; except when the upper lobes are hepatized, when it is sometimes tubular above the mamma. The dulness is rarely so complete and extensive as that produced by copious liquid effusion in the pleura. On the left side too, when the lower part of the lung is hepatized, the stroke-sound is never quite dull, but gets a tympanic character from the subjacent stomach. Gentle percussion gives the best results. If a considerable portion of the lung is hepatized, it may be perceived by the sight or feel, that the corresponding portion of the chest partakes imperfectly in the respiratory movements.

The sputa sometimes continue rusty and glutinous, but more commonly become opaque, or mucous, or watery, or cease altogether. When hepatization takes place, the fever is generally somewhat lowered, and the pulse loses strength; but it remains quick, and is often running or wiry; the dyspnœa is unabated, the face is sometimes patched with red, and in the worst forms shows some lividity. The disease, when extensive, often proves fatal in this stage, without going on to suppuration.

In the third stage the effused lymph in the hepatized lung becomes changed into purulent matter, which is generally consistent at first, but, if life lasts, may acquire the liquidity of common pus.* In this stage the bronchial respiration and vocal resonance usually cease, and are sometimes supplanted by a gurgling mucous rhonchus, indicating the presence of a liquid in the principal bronchial trunks. The expectoration is dirty, mucous, or purulent ; in scorbutic persons it is a brown serous liquid. The pulse now becomes very weak and thready, and the strength generally sinks rapidly.

In the rare case of the formation of an abscess in the hepatized lung, the passage of air through the liquid will be indicated by the gurgling or cavernous rhonchus : and when the cavity has been emptied of the pus by expectoration, pectoriloquy and the cavernous respiration will be added to this sign. Abscesses of the lungs are sometimes gangrenous.

Gangrene is also a rare termination of peripneumony. The gangrenous portion, being softened and ejected by expectoration, will produce a cavity which will be indicated by the usual signs of cavernous rhonchus or resonance. The distinctive physical sign of gangrene is the fetid odour emitted from the diseased part in respiration and cough, and the expectorated matter is also extremely fetid. This change is usually attended by a collapse of the features, and great prostration of the vital powers.†

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* This changes the colour of the diseased lung from the red of hepatization to discoloured yellow, drab, or stone colour, which is frequently mottled with red portions in the second stage, and with the black pulmonary matter. This is called by Laennec *hepatisation grise*, as it retains the weight, granular texture, and compactness of hepatization ; and by Andral *ramollisement gris*, because a slight pressure reduces it to a thick purilage. In a more advanced degree, the colour is of a straw or sulphur yellow, owing to the greater quantity of liquid pus ; the texture is considerably destroyed ; more rarely, in certain parts, it is entirely so, constituting a kind of diffused abscess, which has been known to occupy a considerable portion of the lung. Circumscribed abscess is still more rare.

† The anatomical characters of gangrene of the lung are various. The colour is sometimes like that of a simply engorged lung, with a greenish tint. Sometimes it presents a greenish-brown, or a dark-brown aspect. In its progress the gangrene produces a softening and complete deliquescence of the pulmonary tissue : but the sphacelic fetidity is the characteristic sign. It is sometimes found combined with the suppurative stage, in other cases in the midst of a hepatized lung ; and Chomel thinks that the first stage may, under the septic influence of the effluvia of sewers, pass immediately into a state of gangrenous softening.

Thus far we have traced peripneumony in the changes in the pulmonary textures, as indicated by its pathological signs. The severity of the case may be judged from the extent of the disease, and the advances which it has made.

It is in the first stage of inflammatory injection that auscultation proves pre-eminently useful, in assuring us of the existence of a disease that no other symptoms with certainty discover. The presence of the crepitant rhonchus, together with an inflammatory pulse and hot skin, may be taken as a warning to resort to energetic antiphlogistic measures, which in this stage will seldom fail in arresting the course of the inflammation. The disappearance of the crepitation, and sometimes the presence of the bronchial respiration and resonance, announce the increasing danger and progress of the disease, as they indicate its advance to the second stage. The diseased structure is, however, still susceptible of a return to a healthy state; and the view which we have taken of the morbid anatomy of this stage suggests, in addition to means directed against the inflammatory orgasm, the important advantage with which sorbefacients, such as mercury and tartar emetic, may be used. Occasionally the passage of the disease to the third stage may be inferred by the substitution of a mucous rhonchus for the bronchial respiration; but there is often no sign distinctive of purulent infiltration. The duration of the disease, and the nature of the expectoration, may often lead us to suspect it, and would render our diagnosis proportionately unfavourable. Recoveries have, however, been effected, not only from this state, but even from gangrene, by the sloughing of the dead matter, and the formation of a cavity which has afterwards become cicatrized as an abscess. The chances in favour of this event will be in proportion to the small extent of the disease, and the functional strength of the subject.

The resolution or retrogression of peripneumonic inflammation is attended by a succession of the same physical signs that marked its progress, but in an inverted order. Thus, in a hepatized spot, where no sound of the ingress or egress of air has been heard, or perhaps only a bronchial respiration, a slight crepitant rhonchus begins to be distinguished at the end of each inspiration, apparently produced by the air again gaining a straitened admission through a few of the bronchial tubes, whose calibers have been partially restored by the re-absorption of matter effused around their parietes. This sign increases in intensity as the resolution proceeds; the bronchophony and bronchial respirations are first increased by the free passage of air through them, but subsequently diminished as the lung re-acquires its spongy texture, and becomes a worse conductor of sound.* The sound, on percussion, improves also in like manner. After a while the natural breath-sound sometimes is heard mixed with the crepitant rhonchus, which, from the thinner quality of the bronchial secretion, is subcrepitant, or less dry and regular than in advancing pneumonia, and gradually becomes quite irregular or mucous. As the texture becomes more permeable to the air, the natural breath-sound increases as the rhonchus diminishes, and the healthy function of the lung is thus gradually restored. But here again the signs obtained by auscultation are invaluable, as they alone indicate with certainty the absence of the disease. The dyspnœa may have been removed, the cough may have ceased, the expectoration may have become simply catarrhal, the pulse nearly natural, and all febrile symptoms abated; and yet the auscultator detects the lurking disease in the persistance of the crepitant rhonchus; and as long as this continues, a slight exposure to cold, or an irregularity in regimen, may cause a relapse, which, in a subject already reduced by depletion, may be more difficult to cure than the original disease. The subcrepitant rhonchus, without any attendant signs of fever, often ceases on the exhibition of a mild tonic joined with an expectorant.

I have here described the access, progress, and cure of pneumonia in its general well-marked course. It will now be necessary to advert to certain varieties in the signs produced by the situation, extent, and complication of the pneumonic inflammation.

When the inflammation occupies the central part of the lung, and particularly of the base, without extending to the surface, its signs may be beyond the reach of the ear, or discernible only by a very careful examination; hence the necessity of attending to the sputa and other indications. When, however, the inflammation occupies a considerable portion of the organ, the signs are very evident, and the breath-sound in the healthy parts is much louder than usual, and is called *puerile* or *supplementary*. The intensity of this supplementary respiration will depend besides on the extent of the disease, on the natural activity of the respiratory function, the want of breath, so different (as we have before indicated) in different individuals.

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* According to Laennec, a hepatized lung in progress towards cure becomes paler and moister; if it has been solid for several days, the restoration of the vesicular structure is partial and irregular, and it continues for some time firmer and less elastic than natural. The signs of restoration from the third stage are a removal of the yellow pus, and the substitution of a more liquid serum for the pus. This afterwards becomes drier, and the texture vesicular; but for a long time the lung retains a dirty yellow or greenish colour.

Percussion gives no indication when the inflammation is central; and it requires much practice to detect by it a small circumscribed inflammation on the surface. Its indications are always doubtful at the posterior and lateral margins of the lungs, on account of the vicinity of the abdominal viscera.

There is a variety of pneumonia, called *typhoid*, in which the local symptoms are often very obscure, and the attendant fever is of a typhoid kind; the pulse being very weak, as well as quick, the skin harsh and dry or clammy, the tongue brown and dry, and the urine very scanty and high-coloured. The lungs in such cases are sometimes found after death so engorged that parts sink in water; the texture is soft, and easily broken into a semifluid pulp. In these cases there is dulness on percussion, and absence of breath-sound in the lower and back parts of the chest, but, as Dr. Stokes has remarked, little or no crepitation. This is probably because the engorgement is more congestive than inflammatory, and, being at once produced, renders a great part of the tissue impervious to air. But this degree of congestion, if it occupy the middle parts of the lung, may give bronchophony and bronchial respiration.

The complication of pneumonia with bronchitis is very common. In fact, as I have before said, there is bronchial inflammation in nearly every case of pneumonia. But it sometimes happens that pneumonia becomes added to extensive bronchitis; and this, especially when attended with copious secretion, obscures the physical signs of the pulmonary inflammation. Sonorous, sibilant, and mucous rhonchi are so loud and general, that it is difficult to distinguish whether crepitation be present or not. In such a case we should listen particularly to the end of each inspiration, at the inferior and posterior regions of the chest, about the margins of the lobes, and if crepitation is to be heard at all, it will generally be then and there. We should look out also for the rusty tinge in the sputa, and, as the disease proceeds, for greater dulness on percussion; and we may generally succeed in discovering by these means when the inflammation has extended to the pulmonary plexus of vessels.

The effects of a concomitant pleurisy on the pathology and signs of pneumonia are more remarkable. Whenever the inflammation extends from the lung to the pleura, it may be supposed to increase the serous secretion, and perhaps lead to the effusion of lymph; but when the pulmonary inflammation has existed first, and become extensive, these pleuritic products are commonly to small amount. When the inflammation has simultaneously attacked both the parenchyma and the investing membrane, and nearly to an equal degree, constituting the disease called *pleuro-pneumonia*, the effusion of the pleura by its pressure modifies the effect of the inflammation in the lung. The lung is found after death consolidated, but tougher and redder than in the state of ordinary hepatization, and totally destitute of the granular aspect. It very much resembles the substance of muscle; hence Laennec termed this condition *carnification.* It seems to exhibit the more essential part of inflammation of the lung; the sequel or effect, effusion into the coats of the air-cells, which constitutes granulation, having been prevented by the pressure of the external liquid effusion. This combination is therefore, too, slower in progress than simple pneumonia; the degree of the inflammation, as well as the quantity of its product, is restrained by the external pressure, and it scarcely, if ever, proceeds beyond the second stage. But this slower rate of process tends to make its results more permanent. If false membranes are thrown out on the pleura, they become more firmly organized, and bind down the lung in its more compressed state; and the lymph effused in the tissue itself, scanty though it be in comparison with that of a hepatized lung, may become the means of adhesion of the sides of the compressed cells and finer tubes, and of consequent obliteration of more or less of the proper tissue of the lung. When the liquid effusion is removed by absorption, and when the chest becomes again capable of expansion, what will supply the place of the obliterated cells? There will be the atmospheric pressure at each inspiration brought to act on the obstructed tubes; the air can no longer penetrate to the cells; so it must dilate the tubes, and make their increased size compensate for their defective terminations. Pleuro-pneumonia is, then, as I have before had occasion to remark, a cause of dilatation of the bronchi. I have watched two remarkable cases soon after their inflammatory origin in acute pleuro-pneumonia, through a chronic course, in which signs of permanent consolidation of the lung, dilatation of the bronchi, and contraction of the chest, were the physical conditions which accompanied dyspnœa, weakness, and a dropsical and cachectic state, which ultimately terminated in death, when I found on dissection the conditions which I have described.

On referring to the history of other cases of dilatation of the bronchi, I have met with several in which the symptoms are described to have originated in an inflammatory attack, like pleuro-pneumonia; and I incline much to the opinion, that all the cases in which the dilatation affects the bronchi of one side only, and in which there is also general consolidation of the lung, with some contraction of the chest, owe their existence to this cause.

The signs of pleuro-pneumonia are a combination of those of pneumonia and of liquid effusion in the pleura. At first there is crepitation; but this, as the lung is pushed aside by the liquid, becomes indistinct, whilst the dulness on percussion is much more marked than in pneumonia, at least in the lower parts of the affected side. In the central region of the chest, bronchial respiration and bronchophony are soon produced, by the condensed lung being pushed against the walls; and if a thin layer of liquid intervene, the bronchophony acquires a loud buzzing accompaniment, like the voice of *Punch;* in fact, the voice seems double, which probably depends on a part of its vibrations being modified into a buzzing or bleating, by passing through the thin layer of liquid, whilst other vibrations pass unchanged. The vocal resonance is generally louder in pleuro-pneumonia than in either pleurisy or simple pneumonia; and I suppose this arises from the chief tubes being pressed closely against the walls of the chest, with complete condensation of the vesicular structure. The same circumstance will sometimes give the amphoric or tracheal sound on percussion in the mammary region, which forms a singular contrast to the dulness of other parts: so also I have found the respiration quite tracheal in this spot, and the resonance of the voice quite as loud as that of caverns.

As we have seen, with regard to pleurisy, so in pneumonia, the inflammatory action may not entirely terminate with the effusion of lymph, although it do not lead to the third or suppurative stage. When the acute inflammation is extensive, and the effusion of lymph is not removed by absorption, the disease generally proves fatal before there is time for further change; but in circumscribed peripneumonies, or in small parts of more extensive hepatization, a chronic inflammation sometimes goes on, and produces that kind of tough induration which is the general result of chronic inflammation in a parenchymatous structure. Thus, in the lungs of those who have suffered from long and repeated attacks of inflammation of the chest, even where there are no tubercles, we not unfrequently meet with portions of the tissue which are dense, almost destitute of air and of liquid, tough, and sometimes almost cartilaginous. Their colour varies from a dark dingy-red colour to different lighter shades of reddish-brown and buff, sometimes rendered gray by a mixture of the black pulmonary matter. Their aspect also is varied, like that of acute hepatization, by the tissues that are most affected, it being sometimes granular or oölitic (as Laennec has described it,) from the especial thickening of the individual vesicles; in other cases streaked or veined, from the hypertrophy of the interlobular septa and cellular tissue under the pleura and around the large vessels; in others more uniform, and of a darker colour, from the pulmonary plexus of the vessels being the chief seat of the alteration, and the colouring matter of the blood entering largely into the deposition. In this last variety the cellular tissue between the lobules and under the pleura is somewhat thickened to the amount of several lines, and is of a light drab or gray colour, like that of miliary granulations, and like them has almost the density of cartilage. Now these changes, which thus occur as the sequel of acute pneumonia, are also frequently met with complicated with those states of the lung which are called tuberculous; and a considerable portion of the consolidation that is met with in phthisical lungs, often presents precisely the same anatomical characters as these chronic hepatizations which supervene on acute pneumonia imperfectly subdued. I have also more than once met with them in the lungs of those who have long suffered from extensive organic disease of the heart, where the circulation through the lungs was perpetually impeded by the structural lesions of that organ; and it seems to me that the same mechanical congestion from this cause, which sometimes leads to an effusion of blood in the tissue, constituting pulmonary apoplexy, in other cases, if long enough continued, terminates in an effusion of lymph, and an obliteration and consolidation of the pulmonary tissue. This condition of the lung is sometimes coupled with irregular dilatation of the air-cells; and, on examination after death, the organ presents a knobby surface, and feels nodulated where the consolidation occupies parts or lobules. I think that there is reason to ascribe also, to a minor extent of chronic inflammation of the parenchyma, that increased density and rigidity of the lung, without entire consolidation, which is often found in the lungs of those who have long suffered from dyspnœa.

The signs of chronic peripneumony are those of consolidation and obstruction of the vesicular tissue, which continue after the urgent symptoms of the acute disease have subsided. The dyspnœa has become less oppressive, although it is still felt on exertion; the fever has been reduced, although there is still some quickness of pulse, and a return of heat of skin towards night; there is still some cough; and although there may be improvement in strength and appearance, it is not progressive, the patient remaining with his organs and functions abridged; there is still dulness on percussion, with bronchial voice and breath-sounds in the seat of the late inflammation; and if, in this condition, he neglect the means which may most conduce to the better restoration of his health, the indurated portions of the lung may either prove centres of fresh attacks of acute inflammation, or they may themselves spread, ulcerate, and commence the career of a phthisical disease, which sooner or later will destroy life. I have met with several cases of consumption that have appeared to originate in this manner, independently of any tuberculous disease, or diathesis; the individual having been in excellent health, and quite free from all chest complaints before the attack of acute inflammation, which, afterwards degenerating into this chronic form, laid the foundation of a consumption which ultimately proved fatal. It is, however, much less intractable and slower in its progress than the true tuberculous consumption; for the disease is more local than constitutional; and if circumstances do not occasion its extension, and injure the constitution by a constant and increasing inroad on the functions of respiration and circulation, it may be in the power of nature to effect its removal.

Œdema of the Lung.

Œdema of the lung is effusion of serum into the parenchyma, probably both between the cells and minute tubes, and within them. Its anatomical characters are, that the lung is paler and heavier than usual, pits on pressure, and crepitates little under the finger. It is seldom idiopathic, but, like œdema of other parts, results from some loss of balance in the circulation, an obstruction to the return of blood, or occasionally an excess of exhalation. Thus it may arise from the obstructions occasioned by organic diseases of the heart, lungs, or liver; or from the increased exhalation supervening on exanthematous diseases, particularly scarlatina and rubeola; or on those diseases of the kidneys which interfere with their excretory function, and are accompanied by dropsical effusions in various parts.

Being thus a sequel of other disease rather than a distinct pathological condition in itself, it must vary greatly according to the cause which produces it. When extensive, it occasions dyspnœa, cough, and thin mucous or serous expectoration. The physical signs are a crepitating or *subcrepitant* rhonchus with the breathing, less fine and even than that of pneumonia, and giving proof of the presence of more liquid by the mucous rhonchus in some of the larger tubes. The natural breath-sound is rendered indistinct, especially at the lower and back parts of the chest, where also the stroke-sound is somewhat impaired. Now these signs are very like those of the first stage of pneumonia; and although I have said there is some difference in the character of the crepitation, yet this is scarcely marked enough to constitute a distinction; and a better source of diagnosis is in the general symptoms, the fever, rusty expectoration, and progressive increase of pneumonia being absent, and there being present œdema of other parts, or other signs of disease that may be supposed to produce it. In fact, I should say, from my own observation, that the lungs are less liable to œdema than the external cellular tissue, and that we scarcely ever have œdema of the lungs without anasarca, or at least partial dropsical effusions in the limbs.

PULMONARY HÆMORRHAGE.

Pulmonary hæmorrhage, or *apoplexy of the lungs*, is an effusion of blood into the parenchyma and vesicular structure of the lung. The effusion of blood in the tissue of the lungs may arise from three classes of causes:—1. From circumstances obstructing the passage of blood through the lungs, such as diseases of the heart, especially those impeding the passage of the blood through the left side of the heart, with hypertrophy of the right ventricle; tubercles or other deposits in the lungs, compressing the blood-vessels; violent acts of exertion. 2. From a diseased state of the pulmonary vessels, or parenchyma generally, so that any temporary congestion from exertion, or the continued exercise of the voice, occasions a rupture: in phthisis there is often both this fragile state of the vessels, and an obstructing pressure on them, and the same combination of circumstances produces pulmonary hæmorrhage in some diseases of the heart and arteries. 3. A dissolved or scorbutic state of the blood is another cause to which I should also refer some cases of pulmonary hæmorrhage; for I have several times, in fevers of a petechial kind, met with lungs presenting circumscribed portions of their tissue quite infiltrated with black blood, with effusions of the same kind under the pulmonary pleura, there being no signs of inflammation, and the adjoining tissue being free from disease.*

The symptoms of pulmonary apoplexy or hæmorrhage are none of them constant. There may be dyspnœa, a feeling of tightness or dull pain in the chest, sometimes referred to a particular spot; cough; and what is most common and characteristic, hæmoptysis. The blood expectorated may be only little, and merely tinging the sputa, or it may be coughed up as pure blood; or it may be brought up in large quantities, often containing coagula, by an action like vomiting. The latter kind is generally associated with tuberculous disease, where a vessel of considerable size has been ruptured, and communicates with a bronchial tube. In the case of the hæmorrhagic masses in the parenchyma of the lung, associated with disease of the heart, there is sometimes little or no hæmoptysis, the effused blood coagulating in the tissue before it reaches the larger bronchi. The blood expectorated is generally, but not always, frothy and florid, and mixed with mucous sputa; and it is thus distinguished from that vomited from the stomach, which is more constantly dark coloured and mixed with liquid or other contents of the stomach. I have, however, often seen quite dark coagula coughed up; and when the hæmorrhage subsides, the blood in the sputa acquires a brown tinge. So in examination after death we find the hæmorrhagic spots of some standing, of a deep brownish red colour. Sometimes the expectoration of blood is followed by a relief to the dyspnœa and tightness of the chest; in other instances it is accompanied by feelings of faintness, and where the quantity of blood is large, its effusion may cause extreme danger, and even death by syncope, or suffocation. Or if the immediate effects of loss of blood be borne, there may follow the jarring pulse, palpitation, anhelation, tinnitus aurium, and other symptoms of the reaction or irritation which ensues on excessive inanition of any kind.

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* The effect of these extravasations of blood is to produce patches more or less extensive, of a very dark red or brownish red colour, in the pulmonary tissue, their margins being often quite abrupt. These patches are generally pretty solid, from the coagulation of the blood, and sometimes exhibit somewhat of a granular texture, like that of some kinds of hepatization; but the colours are all much darker. The hæmorrhagic masses may vary in size, from that of a hazel-nut to the whole extent of a lobe of the lung. In the larger masses it is common to observe in the centre grumous or fluid blood, and there is obviously considerable destruction of the tissue. It is not uncommon to find purulent and gangrenous matter in these hæmorrhagic portions; and this is not surprising, seeing that the effusion of blood not only breaks up and destroys the tissue in parts, but also, by its pressure, sometimes obstructs the vessels, and thus destroys the life of the parts which they supply. So, also, we may have hæmorrhagic engorgement associated with pneumonia in any of its stages; for the local obstruction and irritation of a clot of effused blood may be sufficient to determine inflammation of the adjoining tissue; and the distension of the vessels in the first stage of pneumonia may also, in those predisposed to it, be sufficient to occasion pulmonary hæmorrhage. The hæmorrhagic or inflammatory engorgements are likewise sometimes associated together with those congestions which are produced by obstructions to the passage of blood through the heart; so that in the lungs of patients that have lingered for some time with organic disease of the heart, we not uncommonly find apoplectic or hæmorrhagic spots, inflammatory congestion, and hepatization, occurring together.

The physical signs of the effusion of blood in the tissue of the lungs are such as we should expect from the filling up of some portions of the tissue, and the pressure on the parts adjacent. Thus there may be some part of the chest in which may be found dulness on percussion, with absence of breath-sound; and if the consolidation be extensive, there may be bronchial breath and voice-sound. Around this part, which may vary in extent, there may be an irregular crepitation, with a sound of bubbling in larger tubes, arising from the pressure of the coagulated blood on the adjoining texture, and the presence of some in a liquid state in the outer tubes and vesicles. When the disease is extensive, and especially if complicated with tubercles in the lungs, the expansion of the chest will be imperfect on one side. When the foregoing signs present themselves in addition to hæmoptysis, they pretty clearly trace the hæmorrhage to the lungs; but if hæmoptysis be absent, they can only afford a strong suspicion, in addition to the presence of the causes likely to produce it, that there is hæmorrhage or apoplexy in the tissue of the lung. For the dulness on percussion, the bronchial respiration and crepitation, may be equally produced by partial pneumonia; and if there be at the same time bronchitis, the glairy sputa may get a rusty tinge, like those of pneumonia, from the blood effused, without enough admixture of blood itself to constitute hæmoptysis. These constitute difficulties in diagnosis, but the practice required is not equally perplexing.

DILATATION OF THE AIR-CELLS, OR PULMONARY EMPHYSEMA.

Vesicular emphysema is a dilatation of the air-cells of the lungs. It may be partial or general; and the partial kind may be confined to separate vesicles, or it may affect all the vesicles of a lobule; general dilatation may affect one lung, or the whole, or a considerable part of both lungs. The anatomical characters of these lesions present some variety, but they have this in common,—that when examined either through the pleura in the recent state, or after being inflated, dried, and sliced, the air-cells are seen much larger than those of healthy lungs. When the dilatation is general, the pleural surface of the lungs may be as smooth as usual, only more convex; but, when it is partial, either the dilated vesicles or the emphysematous lobules, are seen forming irregular prominences. Individual vesicles are sometimes seen under the pleura, and especially at the margin of the lobes, dilated to the size of a pea, a hazel-nut, and in some cases to a much larger size.

I have noticed another remarkable difference between cases of dilated air-cells, whether general or partial. Some are accompanied with a diminished pliability of the texture of the lung; it has acquired a degree of rigidity; it does not collapse when the chest is opened, and gives a greater resistance to the fingers than a healthy lung does: this kind is generally accompanied by a great deal of the black pulmonary matter. There is here, notwithstanding the dilatation of the air-cells and lightness of the lung, a hypertrophy and toughness of some of its textures; and portions near the root are sometimes found approaching to the dry tough consolidation of chronic pneumonia, but still containing a good deal of air.

The condition of the lung in the other cases is just the opposite of this: the texture is more flaccid and yielding than usual; and when the margin of a lobe is pressed between the finger and thumb, it feels almost like a soft single membrane, it is so thin. It often does not collapse on opening the chest, and this appears to be from a loss of elasticity, for it pits on pressure, like an œdematous lung, and does not recover its shape again; and like it, too, it is commonly much paler than usual. Andral has noticed this condition of the lung under the name of *atrophy*, and observes that it occurs most commonly in old people. I have seen it both general and partial; the partial kind of flaccid dilatation being of common occurrence in the anterior lobules and margins of the lungs in tuberculous phthisis. The fringe of dilated cells, like a row of beads, which is sometimes seen at the margin of the anterior lobes, is also simple dilatation without the least rigidity. On examining the dilated cells, they are generally seen to be smooth, and lined with membrane within, but I have several times found them communicating with one another; so that, on inflating one, the air entered those adjoining. In some instances, the divisions between the cells have been more completely broken down, and reduced to mere threads. They have been sometimes found to contain serum, pus, and even tuberculous matter. These several lesions are frequently associated with diseased conditions of the bronchial tubes, redness and thickening of the mucous membrane, hypertrophy of the longitudinal fibres, dilatation, and partial contraction.

Laennec explained the origin of this lesion in this manner:—In cases of chronic catarrh, particularly of the dry kind, the small bronchial ramifications become so obstructed by the swelling of their membrane, or by the secretion of a viscid mucus, that the air can be forced through them into the vesicles only by an effort. Now, as in ordinary respiration, the inspiration, a muscular effort, is more forcible than expiration, the former may prove sufficient to overcome the obstacle to the introduction of air into the vesicles, while the expiration is inadequate to effect its expulsion. Successive portions of air, expanding by the increased temperature, are thus introduced and incarcerated in the cells, which are thereby kept in a state of continual dilatation. This may be one mode in which the air-cells become dilated, but I think that we shall find other causes still more efficient and common in operation.

When there is partial or complete obstruction in any of the bronchial tubes or cells, the inspired air cannot press with the usual force beyond the obstructions; but it will press with more than usual force into the adjoining tubes and cells, to which its access is quite free, and these latter may thus become distended, and in time permanently dilated. The obstructions to which I allude may be caused by the viscid secretions in the tubes, thickening of the textures, tubercular deposits, and the like; and it is with diseases in which these occur that dilatation of the air-cells is most commonly associated. Tuberculous or other solid deposits obstructing small parts of the vesicular texture chiefly, are especially calculated to cause the dilatation of the cells immediately adjoining; for, rendering inexpansible the parts which they occupy, they cause the force of full inspirations to fall on the pervious tissue; and we accordingly find these deposits almost constantly accompanied by pulmonary emphysema, which makes prominent the lobules or parts of lobules on the surface of the lung after death. Another cause of dilatation of the air-cells, and this of a pretty extensive kind, is rigidity, or want of extensibility of the longitudinal fibres of the bronchi. This change I have already noticed as an effect of chronic bronchitis, and we can readily perceive that if the tubes do not lengthen with the expansion of the chest, the air will press unduly on the peripheral cells, and occasion their dilatation. Thus we find those at the margin of the lower lobes most dilated, for these are most under the influence of the forces expanding the chest. It is not uncommon to find the marginal vesicles dilated in the lungs of old people with ossified cartilages; and I think we must ascribe this also to the comparatively immobile state of the ribs and central portions of the lungs, and the increased action of the diaphragm, and consequent undue pressure of the air into the texture immediately contiguous to this part of the respiratory apparatus. These are the chief mechanical causes by which the vesicular texture of the lung becomes distended; but there are others of a more vital nature, which may also be concerned in the production and perpetuation of this lesion.

M. Andral conceives that the air-cells sometimes become enlarged by a wasting away and breaking down of some of their walls, so that several are reduced to a few of larger size. This may be, doubtless, a cause of the unusually light and flaccid condition of the lungs often found in old people after death; but such a state simply would not constitute the disease which, during life, is so cognizable as vesicular emphysema. I have before said that an unusual flaccidity often attends dilatation of the air-cells, and this probably proceeds sometimes from atrophy, and sometimes from a loss of the proper elasticity of the textures. What we know of the structure and properties of the air-tubes and cells will suggest the existence of another cause of dilatation, in a defective tone of their circular fibres, as well as in a loss of elasticity in the longitudinal. It was observed by Mr. Swan, that in animals which died after the eighth pair of nerves had been divided in the neck, the lungs were found uncommonly distended with air. Again, it was a remark of Laennec, that the lungs of persons who had been asphyxiated by the air of sewers, were always very voluminous, yet filled with air, and he proposes a question, whether this may be an idiopathic dilatation of the air-cells. In fact, if the contraction of the circular fibres be an essential part of natural expiration, it is plain that such influences as those just mentioned, which weaken or destroy their power, must render expiration imperfect. Dr. W. Stokes, of Dublin, has taken this view of the subject, and supposes that paralysis of the bronchial muscles is the chief cause of dilatation of the air-cells. Following the same view which Dr. Abercrombie had advanced with regard to the intestinal muscles, he considers inflammation of the tunics investing the tubes, such as that of bronchitis, to be the cause of this paralysis. I cannot, however, consider the defective action of the bronchial fibres as more than subsidiary in the production of pulmonary emphysema: it may do more in determining dilatation of the tubes; but existence of a contractile power in the vesicular tissue is not proved: my own experiments are opposed to it; and I think that the mechanical causes to which we have been adverting must be viewed as the chief agents in distending this tissue, which is naturally so yielding and ready to adapt itself to a change of pressure.

Reverting to the anatomical differences of the *tense* and the *flaccid* vesicular emphysema, we may well trace in one the effect of an over-active and irregular nutrition of the textures, the common result of repeated or prolonged inflammation: and in the other, the absence of any such process, if not the presence of one of an opposite character, causing a wasting of the same parts. Now it is plain that these opposite conditions will lead to very different effects in the signs and course of the lesion. In the one case, the lungs become comparatively fixed in a distended state; and as they resist the power of the expiratory forces to expel the air from them, unusual exertion is required in inspiration to introduce enough air to serve the purpose of respiration. They are, therefore, perpetually exposed to a distending force; and as the dilatation proceeds, and the increasing rigidity or obstruction with it, the lungs acquire a permanent volume beyond what is usual, even in full inspiration, and they distend the walls of the chest, and press on and even displace the adjoining organs and vessels. Hence may be expected to arise continual oppression of the functions of both respiration and circulation, and cachectic and dropsical disease in the system as the sequel. In flaccid vesicular emphysema, on the other hand, there may be little or no increase of volume of the whole lung, and no pressure on the other contents of the chest. Having arisen from the access of air to the vesicles being more free than the vesicles themselves were capacious, there is not that tendency to accumulation which accompanies the emphysema arising from inflammatory thickening or injured motory power in the tubes. In fact, in flaccid emphysema the tubes are often also dilated, and afford pretty free egress as well as ingress to the air. But the air thus admitted to few dilated cells cannot aërate the blood as it would in many small cells; it is out of proportion to the vessels and blood in the part. Hence, however this kind of emphysema may be caused, it will impair the function of the lung, although it supplies more air to it.

I think that we shall now understand what we have to describe of the symptoms and signs of vesicular emphysema. Inasmuch as it is permanent, it will cause a constant shortness of breath, or dyspnœa; and the least additional obstruction, such as that of a cold, bodily exertion, or flatulent distension of stomach, may increase this symptom to an oppressive degree; so as to resemble an attack of asthma. The permanency of the oppression to the function of respiration, in severe cases, induces a cachectic state of body, which is manifested by pallidity and some emaciation, and a depraved condition of all the excretions. In the tense form of emphysema, with increased volume of the lungs, there are added the symptoms of obstructed circulation, as well as well as imperfect oxygenation of the blood, lividity, and even blueness of the face and lips, dropsical effusions, palpitation, and other signs of hypertrophy of the heart. Dr. Stokes has remarked, that these symptoms are always worse in cases where the lower lobes of the lungs are chiefly affected, which he explains by the greater injury to respiration, which would be caused by the enlarged lung preventing the free play of the diaphragm. The expectoration accompanying emphysema is very various. It is most commonly mixed, a dirty serous fluid, containing portions of tough pearly mucus, or of the opaque sputa of chronic bronchitis. In the attacks of acute bronchitis which frequently occur, it becomes glairy, and often very copious towards their termination, like in pituitous catarrh.

Of the physical signs of pulmonary emphysema, one of the most remarkable is the loud hollow stroke-sound, which is even clearer than that of a healthy chest. This is common to both the tense and flaccid kinds of emphysema; but in the former, when extensive, there is a distinctly raised pitch in the sound of percussion, such as in a less degree can be produced by striking the chest when a very full breath is held in. As in this case, in fact, the walls of the chest are rendered more tense by the increased volume of the lung, and the vibrations which they make are therefore quicker, although, from the elasticity of the contained material, they are still quite free. The increased volume of the lung is manifest in advanced cases, in the shape of the chest, which is unusually convex or rounded. The sides, the front, the back, the intercostal, and even the supra-clavicular spaces, some, or all, present this rounded projection; and when the lower lobes are affected, the heart, the liver, and the spleen, may be displaced by the emphysematous lung, which then yields its clear resonance on percussion over an extended region. In this tense kind of vesicular dilatation, the sound of expiration is prolonged whilst that of inspiration is very imperfect and wheezing, and forms a remarkable contrast to the efforts used to introduce and expel the air. This is perceptible to the eye as well; for it can be seen that, with all the exertions in expiration, the chest is very little diminished, and retains its large convex shape, whilst every supplementary muscle can be seen at work endeavouring vainly to depress the ribs. Is there any wonder that this force, continually exerted on the thoracic vessels through the stuffed lungs, should obstruct the circulation, and cause lividity, cyanosis, dropsical effusion, and ultimately disease of the heart? Inspiration is easier, but even this requires exertion; for breath has to be taken, as it were, on the top of the breath, and even needs the supplementary effort of the cervical and superior dorsal muscles.

There are sometimes heard some odd sounds, which are not those of common wheezing or impeded breathing. Now and then there is a sudden clicking or crackling, as if from the sudden passage of air into, or out of, a set of tubes and cells which were before closed.

The signs of the flaccid form of emphysema may be much modified by the other disease, such as tuberculous deposit, that commonly produces it, but they are essentially distinct from those of tense emphysema; and neither this nor their anatomical difference has been, so far as I know, noticed by authors. There is the clear sound on percussion, but no raising of the pitch of the sound. In old people, where this form of emphysema is uncomplicated with solid deposit, the sound on percussion is clearer and deeper than in any other case. Neither the shape nor the motion of the chest is essentially affected by this kind of emphysema; and the sounds of respiration, instead of being obscure and wheezing, are remarkably loud and even puerile, although they have sometimes a little of the whiffing or bronchial character, from the accompanying dilatation of the tubes. We can understand the reason of all this when we reflect that, in these cases, air passes freely into, and out of, the dilated cells, and that their enlarged size, together with the increased energy which the feeling of dyspnœa gives to the acts of respiration, is a sufficient reason why the sound of both inspiration and expiration should be louder than usual.

Interlobular emphysema is essentially distinct from the preceding affection, although it may be combined with it. It is an effusion of air into the cellular membrane of the interstitial tissues of the lung, and therefore especially between the lobules and under the pleura. It is easily distinguished anatomically, by the air being in the line of the interlobular septa, and contained in angular cells of various shapes and sizes, and not round ones, like those of the lung. Sometimes air is effused under the pulmonary pleura, detaching it from the lung in the form of large bubbles. This affection is commonly produced by violent efforts, or by wounds of the lung; but sometimes from rupture of the air-cells by excessive or sudden dilatation. It may, if extreme, produce sudden, and even fatal, oppression to the breathing; but in slighter cases it is of no consequence, being removed spontaneously. Laennec supposed this affection to cause a sound of rubbing with the motions of respiration, which the projecting emphysematous septa make against the walls of the chest. This is sometimes in successive jerks, so as to resemble the steps of a person mounting and descending a ladder. Subsequent observers have not confirmed this opinion, but have ascribed the rubbing sound to pleural inflammation.

Tuberculous Phthisis.

The last class of diseases of the lungs which I shall consider, are those comprehended under the head *phthisis pulmonalis*, or *pulmonary consumption*. By this name, and more popularly by that of *decline*, is implied a wasting of the body from the effect of a disorganizing process going on in the lungs. In order to preserve that rational connexion which this work is designed to exhibit, between the physical changes and pathological lesions, and the signs and symptoms which they produce, it will be well to give, in the first instance, a brief sketch of the anatomical characters of consumptive disease of the lungs; we shall then be able to trace it in its progress through its various stages and complications, and to comprehend the changes of physical properties which it may induce in the organs of the chest.

When we examine the lungs of persons who have died of consumption, we find them more or less consolidated in irregular masses, and on cutting into them they are generally also excavated in parts into hollows of various sizes, which are either empty or contain a thick liquid matter. On closer examination, and after attentively observing the lungs of many consumptive persons, we are enabled to classify the morbid conditions which they present under the following heads:—

1. On pressing the softer parts of the lung between the fingers, we feel in it a number of hard little bodies, and on cutting into it they are found to be roundish granules, of a light semi-transparent reddish drab or skin colour, sometimes more gray or ash-coloured, more rarely devoid of colour, and quite transparent, of sizes varying from a pin's head to a hemp-seed. Their hardness is considerable, sometimes almost equalling that of cartilage; these are the *miliary granulations* or *miliary tubercles* of Laennec and other writers. They are sometimes found singly studding a tissue otherwise healthy; but more commonly they are in groups of several together, and then they are either clustered in bunches, like berries, or they form a considerable mass, with

the interstitial tissue consolidated and indurated between them. They are commonly isolated in the inferior lobes; in the upper parts, and near the root of the lung, they are usually conglomerated in masses. In the upper parts, too, it is common to find in them opaque specks of a yellowish white colour, which are generally in the centres of the granules, sometimes at their margins. In the distinct granulations the opaque part is little more than a speck; but, in those which form a conglomerated mass, the opacity is sometimes seen extending from granule to granule; and in others it constitutes a mass of granulations.

2. In the next place we find consolidation of another kind, diffused through some extent of the pulmonary tissue, of no particular shape, except that sometimes it seems to be limited to single lobules. In consistence it varies; often it is nearly as hard as the miliary granulations, and in parts it has somewhat of their semi-transparency and colour, but generally it has a darker hue, from the colour of the blood and of the black pulmonary matter in it. The consolidation is pretty complete, and the pulmonary texture cannot be distinguished in it, except here and there the coats of a large blood-vessel, bronchus, or interlobular septum, which are often thickened, and partake of the induration. In other cases the consolidation is less perfect, there being still some air in the tissue, and the adjoining tissue being often emphysematous. In these indurated masses are often to be seen, here and there, more opaque lighter-coloured spots, which are sometimes quite distinct and of a dead yellowish white, like those seen in the miliary granulations. In the lightest and most opaque spots we recognize what we must describe as the third morbid appearance to be met with in phthisical lungs, namely :—

3. Opaque yellowish-white masses of various form and size, generally somewhat rounded. Some of them are nearly as solid as the dark or semi-transparent indurations, but they are much less tough; others have more or less of a cheesy consistence, and some are found in parts approaching to a state of grumous fluidity, still retaining their light colour and opacity. These opaque masses are commonly found within the indurations from which they appear to be formed, and they are just of the same character as the specks before described as occurring in some of the single or aggregated miliary granulations. In fact, as these specks are seen in some in greater numbers and extent, and preceded by an intermediate state of opacity, in parts to per

vade the whole mass, it may be fairly concluded that the clusters and nodules of granulations are also converted into this same opaque friable yellowish-white matter. This matter, which is indisputably entitled to be distinguished as *tuberculous*, is occasionally found also in other situations, unaccompanied by any induration; such as in the interior of dilated vesicles and bronchial tubes, in masses under the pulmonary pleura, and in the bronchial glands. In these instances it is commonly of a friable or cheesy consistence, and has not the hardness which it seems to retain for a while when it has originated in the indurated tissue. But this yellow tuberculous matter, however tough and hard it may be in the first instance, tends to soften, either partially or wholly; and thus the masses are sometimes found consisting of loose clots, in a purilaginous fluid, or wholly reduced into a curdy kind of puriform matter. The tuberculous matter is also not unfrequently found diffused through a considerable extent of the pulmonary texture, constituting the *infiltrated tubercle* of Laennec. In its earlier condition, the lung, in this state, closely resembles the last stage of hepatization, when the opacity which precedes suppuration shows itself. It is very much mottled or marbled, for, besides the yellowish-white opacity which is seen in different degrees in its different parts, there is the black pulmonary matter giving it a gray or greenish colour; there are the whiter coats of vessels and interlobular septa, and spots of red tissue less affected. When the lung in this state is cut or torn, which it commonly may be with facility, its interior presents a granular surface, like that of hepatization, and except that its colour is more varied, and it has generally more of the light opacity of tuberculous matter, it resembles a hepatized lung more nearly than anything else. But in it are seen what are rarely met with in hepatized lungs, circumscribed abscesses or cavities, containing a fluid matter. To this softened and fluid state, then, all the conditions that I have been describing, tend to pass; and they constitute *vomicæ*: when so softened, the matter is evacuated into the bronchial tubes, leaving behind the lesions, which I have next to describe.

4. Lastly, we find cavities or excavations very various in number and form, and of sizes from that of a cherry-stone upwards, to the extent of a whole lobe. Sometimes they contain more or less of the remains of the softened tubercle, or a more liquid pus, or a mixed serous or mucous fluid

tinged with blood, or they may be empty. They communicate with each other and with the bronchial tubes, the process of softening and ulceration having destroyed the terminations of these tubes; but blood-vessels and interlobular septa are often spared by the destructive process, and form cords or bands across the cavities. The blood-vessels are, however, almost always impervious in these cases, and the septa are thickened by depositions of lymph. Their walls are composed of the consolidated tissue of the lung, rough and occasionally sloughy, or of an irregular coat of lymph, or in old cavities of a sort of adventitious membrane, which is sometimes thin, like a mucous membrane, and in some more rigid, and of a fibro-cartilaginous character. When these cavities approach to the pleural surface of the lung, there is often a coating of lymph or false membrane on the pleura at the part, which either thickens it, or unites it by adhesion to the costal pleura. Sometimes, however, there is no such deposit or adhesion; and it occasionally happens that the pleura is also ulcerated, gives way, and being perforated, allows the contents of the cavity, and the air from the bronchi, to pass into the pleural sac, constituting pneumothorax and pleuritic inflammation. There is this remarkable in the position and size of the cavities, that they are almost always largest and most numerous near the summits of the lungs, one or more cavities being often there, when in the inferior lobes there are only scattered indurations. In fact, it may be generally observed of all those lesions connected with phthisis, that they affect the upper and posterior more than the lower and anterior lobes; and that they are also more advanced in the former. This is, however, most remarkable with the circumscribed indurations and tubercles; for with the diffused consolidations, especially of the light opaque kind, (tuberculous infiltration,) the middle and inferior lobes are often also affected, and cavities are more commonly found in every part.

Besides these chief and more essential changes of the lungs in phthisis, many others are often found of a more accidental character, such as hæmorrhagic effusion and consolidation, inflammatory congestion, and hepatization of the lung; products of inflammation in the pleura; inflammation, ulceration, thickening, and dilatation of the bronchial tubes; irregular dilatation of the air-cells, sometimes with rigidity; enlargement and induration of the bronchial gland, with yellow tuberculous matter in its different states in them.

Let us now generalize a little on these changes, which anatomy has discovered to us in the lungs of the consumptive. They may, for the most part, be reduced to two. 1. Consolidation, generally of an indurated kind, and either almost colourless and transparent, or of a pearly gray or reddish drab, or of a dark red or more dingy colour. 2. An opaque yellowish white, parsnip-coloured, friable matter, of various degrees of consistency, being first hard, and afterwards becoming soft, and forming vomicæ: this lighter opaque matter, which is properly called tuberculous, is produced commonly within the consolidations just named, but sometimes elsewhere.

What, then, are these morbid conditions of the lung? What pathological view are we to take of their nature and origin? Laennec considered them "accidental productions, that is, real foreign bodies, which spring up in the substance of the lungs, and may be developed in any other texture of the body." We owe great respect to the name of Laennec, but I must confess that his view of the origin and nature of tubercle has never been satisfactory or even intelligible to me. The only way in which I can interpret it is, that tubercles are parasitical bodies, originating in an unknown way, possessing a life and structure of their own, growing by attracting matter to them, and tending, by their own inherent properties, to go through a certain series of changes. The transparent miliary granulations, the gray miliary tubercles, the gray diffused induration, and a gelatinous infiltration, he looked on all as varieties of these bodies in their first stage, and as all tending *per se* first to become opaque and yellow, then crude tubercle, which is still hard, and ultimately to soften into a cheesy or pasty liquid, which is the mature tubercle.

Now this view involves several assumptions little supported by analogy; for instance, that bodies so different in physical character and texture are the same, and that the stages through which they pass are produced by their inherent properties, and not by the modified properties of the tissue of the organ; and it assumes what has been disproved by observation, that the opaque yellow tubercle is always preceded by the gray or semi-transparent, and that the gray induration must always in time become yellow tubercle, Whilst, then, we admit the accuracy of Laennec's observation, that the gray and semi-transparent indurations tend generally to become yellow tubercle, we must consider his view of the change to be too unsupported and hypothetical to be received as satisfactory.

The view of M. Andral is more simple, and involves fewer assumptions. He considers tubercles generally to be the result of a modified nutrition of the texture, and that they are produced, and go through their changes, through the agency of the vessels of the part, and the blood which circulates them. Although he admits that the miliary and diffused indurations precede the production of yellow tuberculous matter, he supposes them to be, not an early stage of this matter, but the result of chronic inflammation affecting the individual vesicles, or the general texture. The chief peculiarity of this view is, the explanation of the regular form and size of miliary tubercles, by locating them in the individual air-vesicles, just as the same author first accounted for the granulations of a hepatized lung. That the diffused induration, called by Laennec the first stage of tubercle, is the result of chronic inflammation, has been admitted by Chomel and Louis, who otherwise rather incline to Laennec's opinions.

Dr. Carswell (see his admirable "Illustrations of the Elementary Forms of Disease") neither adopts the opinion that the indurations are an early stage of yellow tubercle, nor does he admit that they are more than accidentally connected with it. He supposes yellow tubercle to be a peculiar secretion which takes place especially from mucous membranes, but that it may accompany other secretions, such as that of inspissated mucus in the air-vesicles, or of dense false membranes on the pleura or peritoneum ; and thus he accounts for the gray miliary bodies with specks of yellow tuberculous matter, and the similar admixture of this matter with deposits on serous membranes. But if we examine the miliary bodies minutely, we may be soon satisfied that they contain no inspissated mucus that will account for their increased hardness; the induration is obviously in the texture itself, and not merely contained within the cells: and Dr. Carswell's view throws no light on the manner in which yellow tubercle is produced in the gray or dark indurations, whether miliary or diffused; yet this is a point as well established as any in the pathology of phthisis.

If we examine the induration which commonly precedes the production of yellow tubercle, we find that it differs from the healthy structure certainly in these respects ; that it contains a greatly increased quantity of matter, and that this matter is generally harder than the healthy tissue. Now this increase of substance implies either increased secretion or diminished absorption. That absorption is not diminished in the tissue, is plain from the fact that portions of the healthy tissue are at the same time removed by this process; and that increased secretion is present, is proved by the fact, that the indurated texture presents new characters, and is not a simple accumulation of the matter of the natural tissue. Now, to produce an increase in the nutritive secretion, there must, according to a well-established pathological law, be an increased determination of blood to the part. Does this amount to inflammation? Let us see whether indubitable inflammation ever produces an effect on a simple membrane, like that in question. In treating of pleurisy we found that acute inflammation of the pleura causes an overflow of the nutritive secretion in the form of coagulable lymph, which may soon become highly organized into a soft cellular membrane; but when the inflammation is of a lower and more chronic character, the effused matter is slower in the process of organization, and forms a harder texture of lower vitality—a kind of fibrous or fibro-cartilaginous structure. Will not the same observation apply to the parenchyma of the lung? The overflow of the nutritive secretion caused by acute pneumonia we have found to constitute red hepatization, whether granular or diffused; but when we came to examine the effect of lower and more prolonged inflammation on the tissue of the lung, we had to describe a dark consolidation with increased density, in no essential particular differing from some forms of the indurations which we are now considering. Thus the hard, compact, granular consolidation occurring around excavations, gangrenous as well as tuberculous, and admitted even by Laennec to be the result of chronic inflammation, has occasionally the colour and consistence of the indurations which precede the formations of yellow tubercle; and as I have shown that there is a non-granular form of acute hepatization, so it is reasonable to expect that there may be a diffused or uniform kind of consolidation resulting from chronic inflammation, affecting the interstitial more than the vesicular texture. To such a condition the gray diffused induration, called by Laennec the first stage of tubercle, so exactly answers, that Andral, Chomel, Louis, and Carswell, all concur in considering it to be a chronic form of hepatization. When it is the sequel of the acute disease, or of long-continued pulmonary congestion, there is often much redness in the induration; but where the irritation has been of long continuance, and unattended with the more sthenic degrees of vascular action, or of a very congested state of the lung, the texture is more semi-transparent, dense, and gray, or variously modified by the black pulmonary matter in it. The more uniform colourless masses occasionally present, may be traced to be the interlobular septa, or cellular tissue around the vessels, in a state of indurated hypertrophy. In the bloodless and almost cartilaginous portions, we see the exact characters of the matter of which the miliary granulations, or gray miliary tubercles, are minute samples; and if we adopt the view of Andral, that the regular size of these depends on the chronic induration being located in the coats of individual vesicles, we shall see a sufficient reason for their isolated or clustered characters. Moreover, as we have traced the diffused consolidations of the lung through various gradations, from acute, soft, red hepatization, down to gray induration, so M. Andral has found the miliary bodies presenting the same series of gradation; being sometimes soft and red, in other cases livid and harder, whilst the same lung may contain also the granulations similar in size, but pale or gray, and of various degrees of induration. With regard to the more rare transparent miliary granulations of Bayle, described by Laennec also as an early stage of tubercle, I can state that I have twice, at least, found them on the pleura and on the peritoneum, when other parts of these membranes were coated with dense false membranes, and when there was no trace of yellow tubercle in the body. As to the commoner pale granular deposits on the serous membranes, they are the acknowledged products of chronic inflammation, and their numbers and isolated circumscribed form constitute another point of resemblance to the miliary indurations which, in the lung, pass into the state of yellow tubercle. Without, then, going so far as to assert that the miliary indurations of the pulmonary tissue are always dependent on chronic inflammation, I think we may fairly say that both they and the diffused induration are more akin to the products of this process than to any other that we are acquainted with. The condition of the blood we found to be a material element in determining the products of inflammation in the case of pleurisy; so doubtless it is likewise concerned in the modified nutritive secretions of other textures. The more vital and organizable products are furnished by blood rich in fibrine; and they are easily reabsorbed, or, if organized, are mobile, and sufficiently like the tissues of the part, not to incommode or irritate them. But if the blood be poor in nutrient matter, the deposit from it may be susceptible of only a low degree of organization, and will consequently be not only more difficult of absorption, but also less assimilable to the texture of the part, and more calculated to irritate it as a foreign body. It may thus be seen, that although the lowest degrees of inflammation may be alone capable of producing the chronic indurations, when the blood is healthy, yet, when it is diseased, various degrees of inflammation, nay, even the ordinary nutrient process without inflammation, may be accompanied by the deposition of a lymph of a degraded character, and organizable only into a dense semi-cartilaginous tissue. I shall speak of this again presently.

The semi-transparent gray or dark induration is not always converted into crude yellow tubercle. Sometimes it is the seat of vomicæ, which contains a dirty or bloody pus; and although even in this the curdy matter of tubercle is sometimes seen, it is plain that these vomicæ result from a more direct and speedy process of ulceration or irregular suppuration; another analogous result of continued irritation in the condensed tissue.

And now let us pursue the same mode of inquiry with regard to the opaque pale-yellow tuberculous matter which characterizes the second class of phthisical lesions. Laennec calls this the second stage of tubercle, but neither for its formation nor for its subsequent softening does he assign any other cause than an assumed and unintelligible "inherent property." Dr. Carswell is much more explicit on this point; in fact, all his descriptions of tubercle apply only to this kind of matter. He considers it to be a secretion, *sui generis*, totally destitute of organization—an effete matter, continually separated from blood in an unhealthy state, thrown out chiefly on the free surface of mucous membranes, and producing bad consequences only in proportion as it accumulates in organs, impedes their functions, and acts on them as foreign matter. This opinion, so far as it regards the nature of tuberculous matter, does not differ materially from that long since published by M. Andral, who regards tuberculous matter as a peculiar modification of secretion, more analogous to pus than to any other matter, but materially differing from it. And truly, if we survey the general characters of tuberculous matter, consisting of pale, opaque, albuminous particles, generally deposited in a tissue previously consolidated, and the manner in which it tends to become soft and liquid, forming circumscribed collections like abscesses, or infiltrated through the texture, from which it is expelled like foreign matter, we cannot fail to see some general resemblances to the process of suppuration. We have found that the consolidating lymph of a hepatized lung becomes opaque and light coloured before it softens into pus; but the changes here are too rapid to admit of their being fully watched. But when an analogous process goes on more slowly, and in a simple structure, as in the pleura, we can better trace the resemblance. Thus, in the latent and more protracted forms of pleurisy, we have had occasion to remark that the lymph first effused forms a dense tissue of low vitality, and resembling fibro-cartilage in hardness and colour. If the irritation still continues, this new structure throws out a lymph of still lower vitality, in friable shreds, and, in some instances, in form of a curdy matter, totally incapable of organization, which, mixing with the effused serum, constitutes one kind of empyema. Now such a process in the pulmonary tissue would produce all the changes which we have been describing, in the production successively of gray induration, crude tubercle, and softened tubercle. Thus a portion of this tissue, (whether a single vesicle or part of a lobe,) usually under the influence of chronic inflammation or local congestion, becomes indurated by the effusion of lymph susceptible of a low degree of organization. The original irritation continuing, or the very induration itself determining an increased flow of blood to the part, the new structure evolves in the looser parts of its substance a still less organic form of albuminous matter, a pale opaque curdy substance; but as this cannot be (like that from the pleura) thrown off, it presses on its indurated matrix, and causing its absorption, accumulates in its place: thus is effected the conversion of the gray induration into crude tubercle. This entirely unorganized substance acting as a foreign body on the adjoining tissues and the remains of the living texture within it, causes irritation, ulceration, and the effusion of serum and pus, which, as my friend M. Lombard of Geneva first explained, softens and breaks up the crude tubercle into the curdy grumous matter of the matured tubercle. The same irritation and ulceration gives vent to the matter through one or more bronchial tubes; and thus are formed the tuberculous cavities.

But we have seen that yellow tuberculous matter is produced not only in the gray indurations, whether granular or diffused, but also in softer consolidations, like that of hepatization. It is sometimes seen in rounded circumscribed masses in a hepatized lung; in other instances it pervades with its opaque yellowish gray or mottled colour a whole consolidated lobe. In this, the infiltrated tubercle of Laennec, the gray hepatization of Andral, there are often here and there cavities containing a curdy pus, and communicating with the ulcerated bronchi. There are also occasionally found in it circumscribed indurations and tubercles of older date; but in other instances no other form of chronic lesion is present, and the lung has the appearance of inflammatory engorgement in some parts, of common red hepatization in others, whilst other portions of the same consistence have the opaque yellowish colour of tubercle; and these conditions pass by such imperceptible gradations into each other, that it is impossible to avoid the conclusion that they are parts of the same process. And can we wonder that vessels of the inflamed parenchyma of the lung should at the same time pour out in some parts organizable lymph, in others tuberculous matter, which is the same as albumen, only not organizable—can we wonder, I say, when we see a similar variety produced upon the inflamed pleura, where some portions are covered with good lymph, others with a curdy matter like tubercle, whilst many albuminous particles, also in an unorganizable state, are thrown off with the liquid into the sac? So also, in the very masses of coagulable lymph that an inflamed pleura or peritoneum throws out, there have been sometimes found distinct purulent and tuberculous deposits. And why should we wonder at this either? Lymph, pus, and tubercle, are the same albuminous matter, and differ from each other in mechanical condition and susceptibility of organization, rather than in their chemical nature. According to the microscopie researches of Gendrin, which need further confirmation, lymph is composed of regular globules, which by a vital attraction string themselves into fibres and films, which may become organized, and form membranes. Pus consists of larger and less regular globules suspended in serum; but these globules have no vital attraction for each other, and remain loose, and consequently insusceptible of further organization. Tuberculous matter is wholly devoid of organic form, its particles even not being globular, but irregular, like those of mere dirt or clay, and it must remain where formed, a dead inert mass, until decomposed by chemical agency, or changed by the operation of the surrounding tissues. We can readily perceive that these different properties, although possessed by matter chemically the same, and from the same source, must lead to all that variety of results which we know to follow organizable, purulent, and tuberculous deposits. But the characters of these matters are not always distinct; lymph is not always equally organizable, nor perfectly free from the greenish colour and disintegrating globularity of pus, nor even from the lifeless curdy particles of tubercles; and tuberculous matter often contains flakes or films of imperfect lymph. The diffused tuberculation or infiltration of the lung from inflammation, that we have just been speaking of, generally presents a matter in this transition state. It is neither good organizable lymph, nor is it wholly unorganized tubercle: and the albuminous effusions on serous and mucous surfaces not unfrequently present such an intermediate state, that it is difficult to determine to which class they most belong. *Lymph, pus, and tubercle, pass by imperceptible gradations into each other.* The history of the intermediate products has yet to be more fully studied; and it is a subject of immense importance, for they probably constitute those forms of phthisical lesions which it is most within the power of medicine to control.

But lastly, we have found that tuberculous matter is sometimes deposited in tissues and on surfaces bearing no marks of inflammation or other disease. The structures thus affected are commonly those either very vascular naturally, or peculiarly subject to congestion of blood, such as the bronchial glands, the lungs of children, the spleen of monkeys, &c.; and viewing tuberculous matter as a deposit of unhealthy fibrin from the blood, we see a reason, as Dr. Carswell observes, why it is most likely to be found in those organs where the blood accumulates or passes slowly. Whatever may be the cause which determines the deposition of tuberculous matter in these cases, we know that pus also is sometimes secreted in parts unaffected with inflammation, as in the purulent deposits in the viscera after great surgical operations; and in the profuse discharges of matter from the bronchial membrane, which is found after death to be paler than usual. So, likewise, a matter like tubercle has been met with in the blood itself, within coagula in the spleen and in the heart, and in fibrinous concretions within the blood-vessels. This circumstance tends to show that the fibrinous portions of the blood are liable to be converted into tubercle, independently of any action of the vessels; they lose their vitality, and may in proportion be merely deposited in tissues, or on surfaces, without the presence of any irritation. We are thus again, as in case of the gray indurations, led to trace to the condition of the blood the cause of consumptive diseases of the lung; and it is probably a diseased state of this fluid that constitutes what is called the tuberculous or scrofulous diathesis, in which there is a tendency by vessels in different degrees of activity to deposit tubercle instead of lymph; and when this diseased state exists to a great extent, the tuberculous matter is excreted from the blood without any increased vascular action, but merely as an accompaniment of the natural secretion of a membrane, or instead of the ordinary nutrient deposit of a tissue; and thus it may be either an external or an interstitial deposit. Whatever in such cases determines the first deposition of tubercle in a tissue, will with greater facility effect its growth by the addition of similar matter to a ready formed nucleus. The tendency to the deposit of yellow tubercle, independently of irritation, implies a condition of the blood even more depraved than that which leads, under the same circumstances, to the formation of the chronic indurations; it is an ulterior degradation of the fibrinous nutriment of the tissues, replacing them by a matter not merely inapt in texture and of lower vitality, but wholly destitute of life, and of the principle of organization. When, therefore, tuberculous matter is found in an uninflamed tissue, it may be looked on as a sign of a most decided constitutional taint. In such conditions of the system, tuberculous depositions may take place with great rapidity; and as they are already almost ripe for elimination, the ulceration and destruction of the lung will soon follow; but nothing can give developement to tuberculous disease with such fearful celerity as the occurrence of acute inflammation in the pulmonary tissue. It is from this process in a highly tuberculous constitution, I believe, that the general tuberculous consolidation, called infiltrated tubercle, takes place. The matter deposited is often rather a mixture, or intermediate state, of lymph and tubercle, one product predominating in one part, the other in another;

but it is altogether beyond the reach of the sorbefacient remedies which avail in pneumonia to promote the absorption of simple lymph; and if it do not destroy life by its solid interference with the function of the lung, it speedily runs in many points into softening and suppuration, and the patient is carried off by a *galloping* consumption. In this case the lungs are found more or less solidified, and on incision incipient cavities are seen almost in every part; but there is no induration; the most solid parts have scarcely more substance than a hepatized lung, and they even more readily break down under the fingers.

The developement of the indurations is a work of more time, and their structure makes the destructive process which they induce more tardy; nay, the diffused indurations that form the walls of softened tubercles and vomicæ seem to be a provision of nature towards the limitation of the consuming disease; but, under certain circumstances, even these are formed to such an extent, and so soon, that the patient is destroyed by their first developement. In other cases the first formation of indurations is not sufficient to prove fatal; but as some of these are becoming converted into tubercles and cavities, another eruption or crop of them (as Laennec calls it) takes place, and causes suffocation. I do not conceive, however, that in either of these cases the miliary granulations alone are sufficient to destroy life; but there is commonly with them an effusion of serum or of mucus, which completes the fatal obstruction.

It will be hardly necessary to discuss the question of the seat of the hard gray and the yellow tuberculous deposition. If tubercle be, as we suppose, a degraded condition of the fibrine or nutrient principle of the blood, we may expect it to be deposited wherever the nutritive or the secreting process is carried on—wherever lymph or pus is occasionally found—wherever, in short, blood-vessels run. Instead of being, as supposed by Dr. Carswell, most commonly deposited on free mucous surfaces, its appearance on them is less usual, and bears the small proportion of the liability of these surfaces to the plastic inflammations. Lymph is sometimes, but rarely, secreted by mucous membranes; and it is about as common to find tuberculous matter, or a degenerated fibrin akin to it, in the air-tubes or cells, when the coats of these are entire. It is more common on the serous membranes; but it is much more usually (but not, as Lombard maintains, exclusively) deposited in the interstitial cellular texture of organs, especially those which are highly vascular, and in which blood is apt to stagnate or accumulate. I cannot at all assent to the opinion of Dr. Carswell, that tuberculous matter is, in the early stage of the disease, secreted in equal abundance from all parts of the mucous membrane; and that the only reason why it accumulates sooner and more in the upper lobes, is, that their smaller extent of motion prevents its perfect elimination from those parts. Were this true, how easy would be the diagnosis of consumption in its earliest stage! for every atom of tubercle retained in the upper lobes there would be quantities expectorated from the lower; yet we find that it is very rare to see any expectoration in the earliest stage, except that of a thin transparent phlegm. More probable is the opinion of Broussais, that the upper lobes are the first and most extensive seat of tuberculous change, because the bronchial tubes there are shorter, and inflammation more readily passes along them to the cells. But I apprehend the real reason of their peculiar liability is in the greater abundance of interstitial tissue in them than in lower lobes. The bronchi, instead of being lengthened out into mere membranous tubes before they terminate in cells, divide more immediately and abruptly into short branches and cells, and the delicate vesicular structure is thus mixed up with a good deal of the interstitial cellular tissue which everywhere surrounds the earlier bronchial divisions. The smaller capacity of motion possessed by the upper lobes of the lungs may, too, have a share in disposing them to become the seat of tubercular deposit, not by permitting it to accumulate, but by causing bronchial obstructions to the respiration, favouring those congestions of blood, &c., which, we have seen, are circumstances peculiarly favourable to the formation of tubercles. It is not the yellow tubercle only that predominates particularly in the upper parts of the lung; I should say that it is rather the gray indurations, which become afterwards converted into yellow tubercle. Primary tuberculous deposits are nearly as common in other parts of the lung as at the apex. But if we regard the induration as an organized tissue, formed of albuminous matter of low vitality, but still above that of tubercle, we render its production referable to the same causes as those which engender tubercle, and sufficiently explain the affinity of one lesion to the other.

I have described the ordinary changes of tubercle, from its primary deposition to its softening and evacuation, and the formation of an ulcerous cavern. These caverns become, if life lasts, lined with a deposit of mixed nature, but with an albuminous lymph for its basis; and this is commonly mixed with tuberculous and purulent matter. Hence it seldom adheres long, but is broken up, detached, and expectorated. When the constitutional powers are stronger, and the lung less extensively diseased, the coating of the cavity is susceptible of organization, and in time forms a fibrous or fibro-cartilaginous membrane, which pretty smoothly lines the cavity. If the cavity communicate freely with the bronchi, it will be kept by the pressure of the air from any considerable contraction, to which it naturally tends; and in some instances we have reason to believe that the tubes do become obstructed, and that the contraction of the membrane ensues, and tends to obliterate the cavity. Such contracted cavities are now and then met with, but scarcely ever quite empty; they contain more or less of a pale-coloured plaster-like matter, which consists chiefly of carbonate and phosphate of lime, and sometimes contains earthy concretion. The contraction is evident from the puckering of the pulmonary tissue visible on the pleural surface near the cavity, and the adjoining vesicles are generally dilated to fill up the space. The cretaceous matter is probably secreted by the fibrous false membrane; but it may have been originally more of the character of tubercle or pus, and, being unable to escape, the animal part has been absorbed away, and the earthy insoluble salts are left behind, and accumulate from successive depositions. I have seen a similar matter in the remains of old pleuritic effusions; and perhaps we may associate with it the osseous plates that are deposited in adventitious fibro-cartilaginous membranes, on the pleura, pericardium, and great vessels. This earthy deposit is, however, sometimes connected with an earlier stage of the tubercular formation. I have more than once, in lungs not extensively diseased, met with pale yellowish tubercles, composed of concentric layers of almost cartilaginous hardness: in another part of the same lung these layers are broken or loosened by a plaster-like gritty matter of a calcareous nature; and in another part a whole tubercle may consist of this matter, sometimes containing concretions, and having only a few flakes of albuminous matter in it. This is still more distinctly a specimen of what is commonly called the atheromatous structure, which especially invades the coats of the arteries, and the fibrous parts of the valves of the heart, where it sometimes proceeds to ossification ; and I think that it is to be classed with the gray and yellow tubercle, in so far as it is another variety of matter very low in the scale of organization, and formed of lymph of defective vitality. In these latter cases there is no puckering or trace of contraction about the tubercle until it has evacuated its contents, which it is very slow to do, since it has not the tendency to soften and cause ulceration that makes common scrofulous tubercle so destructive. Hence I have several times seen a few of these tubercles in lungs otherwise healthy, the individuals having died of some other disease.

It will be useful to conclude with a summary of the pathological changes which are produced in the lungs by phthisical disease, and a reference to their analogies to other lesions.

1. Induration, whether granular or diffused, caused by the deposition in the tissue, of animal matter of low organization, under the influence of chronic inflammation, or of defective vitality in that part of the blood which maintains the nutrition of the tissues.

2. The conversion of this induration into crude yellow tubercle, by the substitution of a friable albumen wholly destitute of vitality. This change has its parallel in the increase of fragility and opacity, which precedes the conversion of effused lymph into pus. Sometimes the indurated tissue is destroyed by direct ulceration, without the formation of yellow tubercle.

3. The formation of vomicæ by the softening of crude tubercle, and the original deposition of soft tuberculous matter. These bear analogy to suppuration, and the formation of purulent dépôts.

4. The evacuation of vomicæ, and the formation of ulcerous cavities, which is like that of fistulous ulcers from abscesses.

5. The hardening of tubercle without discharge into cretaceous tubercles ; like the conversion of abscesses into indolent tumours.

6. Accidents contingent on the preceding changes, such as hæmoptysis, from the obstruction or ulceration of blood-vessels, perforation of the pleura, and consequent pleuritic pneumothorax, intercurrent inflammations, bronchitis, pneumonia, ulceration, and other changes in the bronchi, trachea, and larynx.

Besides these, various changes are commonly produced in other parts of the system, such as tuberculous disease in the mesenteric and other lymphatic glands, spleen, liver, brain, ovaries, and other parts; and tubercle combined with induration, or some low form of organized structure on the serous membranes, the pleura, peritoneum, membranes of the brain, &c.; inflammation and ulceration, probably proceeding from tuberculous deposits in various parts of the intestinal mucous membranes; chronic inflammation of the joints, and brown softening of their synovial membranes; diseased nutrition of the interosseous ligaments; greasy degeneration of the liver; and general wasting of all the tissues.

Now, instead of giving a detailed account of the symptoms of phthisis, I shall endeavour to class them according to the pathological stages of the lesions that give rise to them. But before any of these lesions are actually formed, we have found it necessary to assume that there is a cause, either local or constitutional, or partaking of the character of both, which precedes them. So, also, if we attend to different cases of pulmonary consumption, we shall find some in which the individuals had enjoyed very good health until they were attacked with one or more severe colds, or inflammations of the chest, or a fever accompanied by pectoral symptoms, sooner or later after which the phthisical disease commenced. In others, again, the cough and other symptoms begin very gradually, without any very obvious cause, and with as little apparent external reason soon increase to a serious extent, and the consumption runs a rapid career. In a third class of cases, the patients have been out of health, in a debilitated or cachectic state, before the commencement of the cough and other local symptoms, which become suddenly developed after exposure to cold, the stoppage of an habitual evacuation, or some other cause likely to occasion local irritation or plethora.

In the first class of cases we have the developement of phthisis from local inflammation, without any evidence of prior constitutional disorder, unless an hereditary predisposition, which may be traced in some of these cases, may be considered as such. The acute inflammation, whether pulmonic, pleuritic, or bronchial, imperfectly treated and only partially subdued, passes into a chronic form, and either immediately developes phthisical indurations in the lungs, or, by lowering the vital powers generally, leads to their formation from perverted nutrition, or from the irritation of any fresh exciting cause. Under any of these circumstances, chronic inflammation, either by its own local effects, or by its depressing influence on the constitution, or by both combined, becomes a sufficient cause of pulmonary consumption: and, as you may suppose, consumptive disease originating in this way, is sometimes more limited in its extent, and manageable in its course, than that first arising from a constitutional disorder. As the cause is more local, so the lesion is more confined to a part ; and we see instances of it in the very partial indurations or tuberculous depositions which we frequently meet with in the lungs of elderly persons who die of other complaints. In at least half the instances in which I have thoroughly examined the lungs of persons in London and in Paris, above the age of forty, without reference to the disease which caused their death, I have found some traces of those lesions which, occurring in greater extent, constitute phthisical disease. It is easy to discover these by passing the lungs quickly between the fingers, and when feeling a hard body, by cutting into it, it will be found to be either a gray induration or a yellow tubercle, or the remains of one; whilst nothing in the rest of the body betokens any scrofulous taint. Yet the same local causes that produced these solitary tubercles, may engender many; and the greater the number that the local cause thus developes, the more effect will it have on the constitution, which becomes in a manner inoculated by it. Hence, therefore, also, out of a local cause, such as a latent or neglected pneumonia, pleurisy, or peritonitis, may arise a general tuberculous disease, involving, more or less, the whole system. Among the local causes of consumption are to be reckoned, also, the habitual inhalation of fine solid particles, which is contingent on certain occupations, such as those of needle-pointers, dry grinders, stone-masons, miners, colliers, and the like. The dependence of the lesions, in these cases, on the mechanical irritation of the inhaled particles, is sufficiently proved by the presence of these particles in the indurated lung, which in the case of colliers is completely blackened by them. With these cases may be associated those of lesions, resembling those of phthisis, produced in the experiments of Saunders, Cruveilhier, and others, in the lungs of animals, by the injection of mercury into the air-tubes, or into the blood-vessels.

In the second and third class of cases, which are unfortunately the most numerous, the disease is certainly constitutional; and the cachetic condition of the system, as well as the circumstances that seem to occasion it, fully correspond with the pathological views which we have taken of the origin of tubercle. Imperfect nutrition, whether from deficient or improper food, or from a permanently disordered state of the digestive or assimilative organs; unhealthy air, whether from closeness, humidity, or impurities; long continued exposure to cold (as from insufficient clothing) not intense enough to produce inflammation; depressing passions, such as disappointed love, anxiety or distress from reverses of fortune, or other severe calamity; venereal excesses; profuse and very weakening discharges; adynamic fevers; irregularities of the uterine function, especially those that lead to chlorosis; the sudden suppression of habitual discharges, or of long-established cutaneous eruptions, —these, singly or combined, are the most common causes of the constitutional origin of tuberculous matter: they all tend to destroy the balance of the functions, and diminish the tone of the system, and with it that rich fibrinous and vital condition of the blood, by which proper nutrition and the organic functions are sustained. The impoverished blood, defective in that vital albumen with which the tissues are fed and renewed, deposits in its stead a degraded matter, imperfectly or not at all organizable, like that resulting from the lower degrees of local irritation or inflammation. The lungs, the lymphatic glands, aud a few other organs, become the first seat of these deposits, because their textures are in freer relation with the blood than those of other parts are; and if there be in these organs also a congestion, an irritation, or an inflammation, the deposition becomes more extensive and rapid in proportion, although, if the inflammation be acute, it may also produce organizable lymph, together with the degraded albumen, tubercle. And mark how the natural activity of the nutritive process will determine the rapidity of the tuberculous deposition, and the progress of the disease. In young persons nutrition is most active; so is consumption spontaneous and rapid; and at no period is this more manifest than between the ages of eighteen and twenty-five, when growth becomes completed, yet the vessels and their blood do not immediately lose their habit of deposition. But throughout the whole period of childhood and youth tuberculous disease is very common, and is more easily engendered than when nutrition is less active, under the influence of the causes which I have enumerated. We can understand, too, why this morbid nutritive activity, this disposition to deposit albuminous matter, (in such cases inorganic, tuberculous,) should be shown in women after the completion of utero-gestation, and in persons on the speedy healing of large suppurating wounds—circumstances which, as long as they continue, are known often to suspend the progress of consumptive disease. For a further account of the constitutional causes of tuberculous disease, and for the only good description of the condition of the system which precedes its developement, I must refer to the work of Sir James Clark on Pulmonary Consumption and Scrofula, by far the most complete work which we possess on the subject.

Let us now quickly enumerate the symptoms of pulmonary consumption in the order of the stages of the lesions which produce them.

First stage.—The indurations are accompanied as well as preceded by various irritations, both local and general. Of the local irritations one of the earliest is *cough*, which at first is generally slight, and merely hacking, but more or less constant; either dry, or attended merely with a thin transparent mucous expectoration. Another occasional sign of local irritation is *pain* in the chest, which is commonly referred to the sternum or clavicles, and is rather a soreness, or an unusual sensibility to cold or exertion, than a continual pain. Of the more general irritations, *quickness of the pulse* is the most constant, but it is not universal. The quickness is frequently not uniform at first, but depends on trifling causes of excitement, and the pulse may be very slow and weak in the intervals; but as the organic lesion increases, it gradually becomes more constant,* and is accompanied by an irritated state of other functions—a general febrile state. But even then there is not power enough in the circulation to maintain a general or constantly increasing heat; it is manifested most towards night, after the accumulated excitements of the day; and it is generally most felt by flushing of the face, and heat in the palms of the hands and soles of the feet, where the thickness and hardness of the cuticle prevent the perspiration and evaporation which moderate the temperature of other parts. Like other weak febrile movements, this terminates by perspiration more or less profuse, which, occurring in the night, leaves the pulse lowered, but the frame weakened and exhausted in the morning. It is only in the severer cases that this general irritation, or *hectic fever* as it is termed, becomes marked at this early stage of the disease. There is often also gastric irritation, with a white tongue red at the edges, thirst, costive bowels, and turbid urine. These symptoms are perhaps more remarkable in this than in the after stages, when the irritation is more confined to the organs of respiration and circulation.

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* Dr. Guy has ascertained that the pulse in phthisis is much less affected by change of posture than in health. This would add a valuable element of diagnosis; but, from a few observations, I should fear that it is applicable rather to the confirmed than to the earliest stage of phthisis.

The symptoms from *obstruction* comprehend those from the *obstruction to the air*, *to the blood*, and *to the motion of the lungs.* The indurations, granular and diffused, when extensive, *by obstructing the passage of air* to the lungs, occasion the short breath, felt at first only on exertion, so common even in the early stage of consumption. Nay, cases happen in which an abundant formation of miliary tubercles, together with the œdema or bronchorrhœa that they excite, prove fatal in their first stage, by the obstruction which they cause to the passage of air. We have seen that tubercles, or any other partial obstructions, may become a cause of pulmonary emphysema, or dilatation of the air-cells, in the manner which I have already explained. The indurations, by *obstructing the blood-vessels*, give rise to many formidable pathological effects. They may thus cause sanguineous congestion, hæmorrhage, inflammation, œdema, gangrene, and atrophy of the pulmonary texture, hæmoptysis, profuse bronchial secretions, effusion into the pleura, disease of the heart, and so forth; and the symptoms which these lesions produce may be variously grouped in the history of different cases of phthisis. The hæmoptysis occurring in the early stages of consumption is generally from this cause; and it is a serious symptom, not only because it may endanger life by loss of blood or direct suffocation, but also because it is often accompanied by hæmorrhagic consolidation and rupture of the textures of the lung, which tend to accelerate the disorganizing process, and promote the further deposition of tubercle. In some instances, however, hæmoptysis is followed by decided relief to the dyspnœa and cough, having removed a congested state of the vessels. The *obstruction to the motions of the lungs* may be caused by the same circumstances that impede the free admission of air to them, but, in case of extensive solid deposition, may also result from their mechanical resistance to the respiratory movements; and this not only constitutes a physical sign which we shall consider afterwards, but it also keeps the intervening unaffected texture in a fixed state, liable to consequent congestion and further deposit, and adding further to the incapacity of the organs. When once the integrity of a nicely adjusted apparatus like that of respiration is extensively injured, we see how the mischief becomes extended: disorder begets disorder; and unless the counteracting or reparatory powers come soon into action—unless the indurations are diminished, or the vessels blocked up—the whole of that part of the lung may soon become a solid mass. Thus, I believe, sometimes arise those extensive masses of induration that are so commonly met with in the superior parts of the lung.

Second stage.—On the conversion of the gray or dark-red indurations into crude yellow tubercle, and the original deposition of this matter, in addition to the symptoms of irritation and obstruction, which still continue, there are indications of an increasing cachexia, a depression of the functions. The pulse loses strength; the fever is of shorter duration; and the sweats are more profuse; except at times of excitement, the colour of the cheek fades, or is reduced to the circumscribed hectic patch; the expectoration becomes more abundant, or less thin and transparent, and particles of curdy or cheesy matter can sometimes be detected in it. But all these symptoms more properly and fully belong to the

Third stage, in which the tubercles become soft, partially or entirely liquid, and are evacuated by the aid of the secretion and ulceration of the adjoining textures. Then comes on, in addition to the other symptoms, the copious and heterogeneous expectoration of pus, mucus, softened and occasionally solid tubercle, blood, shreds of lymph, rarely portions of pulmonary tissue, sometimes very fœtid. Then follow the usual constitutional concomitants of extensive unhealthy suppurating ulcers, confirmed hectic and marasmus, rendered here more pronounced by the organ affected, and the relations which it bears to the process of sanguification. Then are the dyspnœa and cough increased by the continual discharge of matter into the air-passages, and by the extension of the diseased depositions and ulcerations of the tissue. Yet it is a curious circumstance, that these symptoms are often inconsiderable in proportion to the terrible extent of the organic mischief that has been produced: the dyspnœa is not painful, like that of asthma; it is a state of breathlessness, rather than of distressing oppression; the cough is not so violent as in chronic bronchitis; the pain may be but slight, or none at all; the countenance, though thinned, hectic, and with the sharpened nostrils habitually moving at every breath, may have a clearness in it, with a colour in the lips and a brightness in the eye, which are never seen in other diseases; and the frame of mind is often in the same unconscious and hopeful state, indicating a degree of freedom from those painful struggles in which the vital powers commonly contend with other serious disorders. Now I apprehend the chief reason of this exemption from suffering lies in a sort of balance that is produced among the injured functions. The available part of the lungs is reduced to a surprising extent; but so is the mass of blood that has to pass through it. The free expectoration and the colliquative discharges are continually bringing down the bulk of the circulating fluids to the lessening capacity of the remaining lung. The night-sweats especially are a periodic discharge of the amount of fluid which is beyond what the reduced system of blood-vessels can quietly hold; they generally cease when the fluid ingesta are carefully reduced. So the secondary pulmonary irritations, congestions, and inflammations, are continually relieved by the purulent expectoration: it is a safety-valve which gives vent to these before they can cause distress; and although the destructive process is here perpetually proceeding, the lungs are decaying, the body wasting, and the strength failing, yet it is all by even degrees, a *facilis descensus*; the thread of life dwindles away, fibre by fibre, without struggle or shock; and gentle is the parting of the last filament, when the body drops to earth and the soul rises to eternity.

But the progress of consumptive disease is not always thus painless and unharassed: the sufferings from dyspnœa, cough, pain, heat, and feelings of extreme weakness and sinking, are sometimes very severe. There are those whose animal sensibility is more acute than their organic life is active: to such persons any disorder is distressing; and, even in consumption, the cough, the pains in the chest and sides, the alternate chills and heats, the oppression of dyspnœa, the faintness of debility, besides innumerable pains and aches of various parts of the body, are perpetual sources of complaint. But without an unusual degree of sensibility in the subject, the course of consumptive disease may be rendered rough and painful by what may be called the accidents or irregularities contingent upon it. Intercurrent congestions, hæmorrhages, and inflammations, taking place in the lungs or their investing membranes, may give rise to symptoms of these several acute lesions, superadded to those of phthisis. I have said that a free expectoration tends to prevent these accidental complications; and, accordingly, their occurrence is often preceded or accompanied by a suppression of this discharge, or an alteration in its quality.* But there is an accident which especially tends to ruffle and hasten the course even of the quietest forms of consumptive disease: this is perforation of the pleura, and the consequent pneumothorax and acute pleurisy which it produces. I think that perforation of the pleura occurs generally in cases where the constitution is decidedly tuberculous. It implies a want of that self-preserving energy by which the mischief of ulceration is limited by the timely effusion of plastic lymph. This is a part of the nutrient function, and I suppose this is commonly more active in women than in men; for if I can judge by my own experience, perforation of the lung very rarely occurs in females: I have never met with one instance, yet I have seen nearly thirty cases in the male sex; and, so far as I can recollect, the cases recorded by authors are chiefly of that sex.

Other symptoms unconnected with the chest occasionally attend pulmonary consumption. The larynx is very often diseased in phthisis; the hoarseness or loss of voice being an early symptom, and taking attention from the real and chief seat of disease. The lining membranes of the larynx, and the vocal ligaments, are in such cases thickened, eroded, and ulcerated, and the cartilages and bones may ultimately become carious or necrosed. There is even in these lesions something of the intractable character of tuberculous ulceration; we should therefore beware of *laryngeal phthisis*, for if it be not already complicated with tubercles in the lungs, they may be formed at any time, for the leaven is in the system. The disorder of the digestive organs, which, in the earliest (the irritative) stage, had something of the gastritic character, with red-tipped tongue, thirst, and indigestion, perhaps some tenderness in the epigastrium, commonly passes off when the pulmonary irritation is relieved by the discharge. At this period, however, the bowels often become disordered, and constipation and diarrhœa alternately prevail, dependent on inflammation and ulceration, often complicated with tuberculous depositions in the follicular structure of the mucous membrane of the intestines. The mesenteric glands, too, become frequently involved in similar disease; and thus arise additional causes of exhaustion and atrophy in the colliquative discharges, and the obstruction to nutrition that ensue. The character of the alvine secretions sometimes shows a deficiency of bile; but this is a symptom which more frequently precedes phthisis than accompanies it. Sometimes there are great tenderness and even pain in the abdomen during the whole course of the disease, with occasional exacerbations: these symptoms generally depend on granular or tuberculous depositions on the peritoneum, combined occasionally with inflammation of the membrane. More rarely, tubercles occur in the brain or spinal marrow, or their membranes, and cause symptoms of mental disorder, convulsions, or paralysis.

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* Dr. Stokes has made similar observations with respect to the influence of expectoration in relieving the local symptoms of phthisis.

Let us now pass on to the consideration of the physical signs of pulmonary consumption, tracing them in the different stages of the textural lesions which we have described. In proportion as these lesions are of great or small amount, or are concentrated within a small space, or are scattered widely through the lung, they will produce more or less appreciable signs. Thus the miliary indurations, even in considerable number, may be scattered through the tissue of the lung without producing any distinct diminution or change in the respiratory sound, or in the resonance of the chest on percussion. Sometimes there is a general submucous or subcrepitant rhonchus; but this proceeds less from the tubercles than from the secretion which their irritation causes in the adjoining bronchial tubes: it is the sign of a partial bronchitis, therefore, and can only be taken in evidence of the probable presence of tubercles, when it continues permanently day after day, instead of tending to become sibilant, and to pass off as the rhonchi of common bronchitis do. But it seldom happens that even the early indurations are equally scattered through both lungs. Their tendency is to accumulate in greater numbers or in little clusters near the apices of the lungs, and generally more on one side than on the other. Here, then, will be a concentration of effect, and a difference between the two sides of the chest; and if we explore the corresponding region on the exterior, which are the clavicles and the space immediately beneath them, and the upper ridge of the scapulæ, we may find differences in the sound on percussion, or those of respiration, and the voice, which, according to known principles, may be interpreted as signs.

The clavicle on one side, when struck lightly downwards on its middle, yields a sound duller than that on the other; and especially if this difference extends to the other parts just mentioned, it is exceedingly probable that there is tubercular consolidation of the lung in that part. Great care must be taken to strike both clavicles at the same point, or both infra-clavicular spaces in the same mode, or the comparison will not be a fair one. To avoid error, the parts should be quite uncovered, and the finger should strike on corresponding points of both clavicles. Below these bones, gentle percussion with the flat fingers, mediate or immediate, generally succeeds best; but various kinds of percussion should be tried in doubtful cases, and in different stages of the respiratory act—on a full breath, and after exhausting the lungs. The consolidations may be merely granular and scattered, and they may not have body enough to affect the vibration of the walls, if the stroke of percussion be hard: nay, sometimes the gentlest possible *patting* of the space under the clavicles is the only mode in which any difference can be detected. In the posterior region, however, and above, on the scapular ridge, some force is required to get any pectoral sound at all, and this must be used by mediate percussion on a finger laid along it; comparing, as usual, the two sides. We should always seek for differences in the sound where differences ought not to exist; and the mode, place, and force of percussion should be varied at different times, but be carefully the same in each act of comparison. When the infra-clavian spaces fail to give any signs of disease, the parts below, at the sides and in the back, should be tried. Between the scapulæ is not an unfrequent seat of dulness, especially in children, where the disease occupies the bronchial glands.

We should not omit to use our eyes, also, in scrutinizing the condition of the chest, by making the patient breathe fully whilst we stand in front, in a good light, and watch and feel its motions. It requires more consolidation than is usual, in the doubtful stages of the disease, to produce any considerable irregularity in the shape or movements of the chest; but we can often perceive a slight difference between the two sides: the upper ribs do not move quite so much on one side as on the other. It has been also noticed by Dr. Stokes that a slight flattening or hollowing under the clavicles sometimes occurs in the earliest stages of phthisis. M. Fournet ascribes this to fibro-cellular adhesions, rather than to the miliary tubercles themselves. I have seen some facts which confirm this opinion.

The stethoscopic signs are more delicate, and perhaps more equivocal, than those of percussion. The indurations may form slight partial obstructions to the passage of air, and thus cause a permanent slight wheezing, whistling, or roughness in the breath-sound, particularly in expiration, not removed by full inspiration or cough. If more numerous or extensive, they may transmit the sound of whiffing bronchial breathing in parts where naturally the vesicular only is heard, whilst the soft vesicular breathing is impaired in its force. The sound of expiration is unusally audible, and sometimes equals that of the weakened inspiration, which is almost the only sound in purely vesicular respiration. When, as it often happens, the partial indurations are accompanied by a dilated or emphysematous state of the neighbouring air-cells, the sound on percussion will be less altered than usual; but the breathing will be more whiffing, or more obscure, according as the dilated cells are more flaccid or more rigid than usual. When more rigid, they diminish not only the sound of respiration, but also the motion. The sound of the voice is transmitted by the indurations in an unusual degree, being sometimes only a diffused resonance, but sometimes quite a little voice under the clavicle; but without the articulation of the oral voice. So also the sounds are occasionally transmitted with uncommon clearness from the subjacent arteries, being either double from the second sound of the heart, or single from the mere impulse; and now and then the single pulse is accompanied by a whizzing or blowing, confined to that part indicative of partial obstruction of the subclavian artery, from the pressure of the indurations at the apex of the lung.* I have known it to intermit regularly, occurring only in two or three pulses during the first part of expiration.

Now most of these stethoscopic signs derive their importance directly from the situation in which they are heard, and from their comparison with other parts. There are often some bronchial voice and breath sounds, naturally, near the sternum, between the scapulæ and in the axillæ; therefore we must not place confidence in such signs in that neighbourhood, unless they be more distinct on one side than on the other; or accompanied by dulness on percussion. We may trust to them more towards the humeral end of the clavicle; the angle formed by this bone and that of the shoulder is the proper stethoscopic corner, and the signs heard there are the most unequivocal; but even here a permanent discrepancy between the two sides gives the surest indication of disease; for there are many modifications of the natural sounds heard there, especially those of the voice.

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* This subclavian arterial murmur has been noticed by Dr. Stokes as a sign of incipient phthisis; but it is a very uncertain one, for I have met with it in cases where there was no reason to believe tubercles were present.

This is a very difficult point of physical diagnosis, and requires much experience and nice perception on the part of the auscultator.* But we are not on this account to be dis-

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* Every faithful attempt to render the diagnosis of the early stage of phthisis more certain and precise deserves attention; but when this attempt is founded on very refined distinctions, and on differences between phenomena that are themselves with difficulty recognised, it is likely to be curious rather than useful; and however well the original observer may be convinced of their reality, the distinctions will scarcely be used or recognised by others. These remarks seem to me to be quite applicable to the attempt of M. Fournet. To give the reader a specimen, I extract from the British and Foreign Medical Review (April 1840) a tabular view of M. Fournet's three *phases* of the signs of the first stage of phthisis.

First Phasis. A few small tubercles scattered through the lungs.	Inspiration,	Dry, rough. Intensity increases to 12 (10 natural). Duration falls (from 10) to 9, 8. Quality natural.
	Expiration,	Dry, rough. Intensity, Duration, } Rise gradually to 8 (from 2) Quality, clear ringing.
	Commencing bronchophony in rare cases.	
Second Phasis. Infiltration of crude tubercles in groups.	Pulmonary crumpling sound.	
	Dry crackling rhonchus.	
	Sonorous, sibilant rhonchi (bronchitic).	
	Inspiration,—	intensity = 12, 14. Duration = 9, 8. Quality, clear ringing.
	Expiration,—	intensity, Duration } = 6, 10. Quality, blowing, rarely bronchial.
	Dryness and roughness of respiratory murmurs now masked by change of quality.	
	Slight bronchophony, frequently.	
	Slight obscurity of sound on percussion.	
	Diminished vocal fremitus.	
	Unnaturally distinct transmission of the heart-sound.	

couraged from attempting it; on the contrary, it the more deserves and requires study; and if the student will only devote proper attention to the method, and take every opportunity of practising it, he may in time acquire the power of detecting phthisical disease in its earliest and least intractable stage, and he may thus be enabled to save many valuable lives from otherwise inevitable destruction. He must not neglect attention to the general symptoms; for besides the assistance which they give in the diagnosis by the character of the cough and pulse, they are of the utmost importance in guiding the practice, which is to be adapted to the state of the whole constitution as indicated by them, as well as to the local disease, which the physical signs alone may have discovered.

The conversion of the semi-transparent, gray, or dark consolidation of the lung, into crude yellow tubercle, is a point only deduced from anatomical examinations. We have no certain signs of this change during life. I have before observed, that there is sometimes an abatement of the symptoms of irritation, which may perhaps be associated with this change; there may be at the same time an increase in the expectoration, and the submucous and mucous rhonchi become more marked. But the change to yellow tubercle can hardly take place without some augmentation of the consolidation; the indurations increase in extent, and some yellow tubercle may perhaps be deposited or secreted in other parts. Hence there is a fuller developement of the signs of an increased density in the lung: the partial dulness on percussion becomes more marked; the breath-sound becomes more obscure or more bronchial, with loud expiration, and it may be accompanied by a permanent fine

Third Phasis, or of transition from first to second stage.	Humid crackling rhonchus.
	Sonorous sibilant rhonchi, as before.
	Pulmonary crumpling sound disappears.
	Inspiration,—intensity = 15, 18.
	Duration = 7, 6, 5.
	Quality, blowing, or slightly bronchial.
	Expiration,—intensity, Duration = 12, 15, 18, 20.
	Quality, bronchial.
	Strong bronchophony or imperfect pectoriloquy.
Commencing softening.	Sound more obscure, or even dull.
	Vocal and tussive fremitus much diminished.
	Diminution of partial movement of ribs, corresponding to indurated mass.
	Transverse retraction of corresponding part of the chest.
	Subclavicular flattening.

bubbling or crepitation. The vocal resonance may have also increased in degree and extent, and altogether the signs have become more localised, and therefore less equivocal.

But it is the softening and evacuation of tuberculous matter that produce the most remarkable and cognizable changes in the physical signs; and these too, often give to the expectoration something of the precision of a physical sign. The sputa before may have been sometimes opaque and muco-purulent, as in bronchitis; but they now become decidedly purulent, often sink in water, and, if narrowly examined, may sometimes be found to contain particles of a curdy or clotted substance, like cheese softened in water, which is tuberculous matter; they are not fœtid like the similar concretions from the tonsils. There may also be little streaks or even clots of blood; but this is uncertain: there is generally, besides, more or less mucus, which gives tenacity to parts of the expectorated matter; but on close examination it may be seen that some sputa are opaque purulent clots, almost without mucus; and it is these that come directly from the cavities. Wherever these changes take place, generally under one of the clavicles, or above the spine of one of the scapulæ, there may be heard a clicking or bubbling sound, which is coarser, and gives the idea of its being produced in a larger space than any of the common sounds of these parts. And this sign is more conclusive, the finer and more completely vesicular is the natural structure of the lung in the part in which it is best heard. In listening for this sound, we must look for it in a long inspiration, or during a cough, which makes the air enter more fully and suddenly; and at first we may hear no more than one or two *clicks*, from the entry of single bubbles; but as the evacuation of the softened matter proceeds, and there is more room for the entrance of air, there is then a more continued gurgling or bubbling sound; and this will be coarse and distinct in proportion to the extent of the vomica, and its communication with the air-tubes. This gurgling or *cavernous rhonchus* will also vary somewhat, according to the quantity and liquidity of the contents of the cavity; becoming less crackling and more blowing as these diminish. When it is heard over an extended space, there are probably several cavities communicating with each other, and all containing more or less liquid. It is not necessary to detail all the varieties that circumstances may produce; when we know the principles of the phenomena, we can foresee and understand their varieties.

The softening and evacuation of the vomica being complete, there is left an ulcerous cavity or cavern, which becomes the seat of further phenomena. Even before all the liquid is evacuated, we sometimes hear, in the corresponding part of the chest, with the gurgling a hollow whiffing or blowing sound; and when the patient speaks, a sort of *snuffling* voice, interrupted, broken up by the gurgling. When the cavern is empty, these pass into *cavernous respiration* and *pectoriloquy.* Cavernous respiration is something like what is heard on applying the stethoscope to the front of the neck, in the course of the windpipe; but it is more circumscribed, and does not give the same impression of a rush of air. We may get a better imitation of it by blowing into thimbles or shells of different sizes. It may present considerable variety, according to the size and shape of the cavity, and the freedom with which the air passes into and out of it from the bronchi. When of very large extent, the sound becomes amphoric, like that produced by blowing into an empty phial, and precisely on the same principle. All these phenomena are better obtained with quick forcible respiration, or slight coughing, which increases the force and velocity of the passing air, and exaggerates all the sounds.

Pectoriloquy is another very striking sign of a cavity in the lungs. Its value was perhaps overrated by Laennec; but I think it has been neither appreciated nor understood by subsequent writers. I formerly explained that the voice, although formed in the larynx, vibrates in full strength through the windwipe and its branches, until it becomes broken up and muffled in the smaller tubes and porous tissue of the lung. But if a cavity be formed in this parenchyma, communicating freely with the tubes in which the voice is strong, it will form a part of those tubes, and the vibrations will be continued in *system* from them to it; and there may thus be heard near the surface of the lung a voice from the chest like that heard over the trachea; its distinctness and intensity, however, being more or less perfect according as the cavity is adapted to receive the vocal resonance from the tubes, and to transmit it to the walls of the chest. Laennec made an artificial distinction between the degrees of pectoriloquy, according to whether the voice does or does not give the impression of passing up the stethoscope with the stopper to the ear. In the *perfect* kind, the words are so distinct that it seems as if the patient had his mouth to the tube; where this impression is not produced, the pectoriloquy is *imperfect*. But this is only a difference of degree, the sound most perfectly transmitted *engrossing* the air in the tube with its vibrations; whereas a less perfect transmission affects it more partially. I consider the *character of the sound*, and its *circumscribed position*, a better distinction. The sound is not a mere vocal resonance, like the bronchophony from consolidation, which is often as loud or louder, and may pass up the tube; but it is an articulate although indistinct speaking, and sometimes accompanies a loud whisper as well as vocal utterance. There is in it another feature which is characteristic, and distinguishes it from bronchophony; it is accompanied or followed either by whiffs of cavernous respiration, which give the pectoriloquy a *snuffling* character, or by a hollow or fistular resonance, like that which is produced on speaking at the mouth of the tube of a pan-pipe, the pipe of a large key, a shell, or any such hollow body. This accompaniment is sometimes observed when the pectoriloquy, or transmission of the articulate voice, is very imperfect, so that the voice in the spot sounds like a whisper; but I have found it to be more distinctive of a cavity than the loudest vocal sound without it. I suppose that it depends on the same physical cause as the phenomena of the hollow bodies to which I have compared it; the cavity in the lungs being in the same relation to the bronchial voice as they are to the oral voice. When the cavity is very large, the accompaniment is more amphoric or bottle-like; and if the communication with the bronchi be at the same time narrow, the voice may scarcely be transmitted to it, but only excite in it a tinkling echo, a metallic tinkling, as in pneumothorax. All these hollow, fistular, or tinkling characters, may be also perceived in the breathing and cough, especially in the latter, but not in a proportionate degree; and I have known them to be scarcely perceptible but with the voice. These differences must depend on the relations of the cavity to the air-tubes communicating with it; if this open into them so as to catch the current of air passing in them, its interior will be thrown into vibration; otherwise the air within it may only receive the stronger and more pervading vibrations of the voice. So also, if there be much consolidation about and beyond the cavity, there may be very little passage of air in the tubes, and therefore but little cavernous breathing.

The circumscription of pectoriloquy is another of its peculiar characters, and by this it may generally be distinguished from the loud bronchophony of condensed lungs, which is diffused over some extent of surface. To observe these differences, it is quite necessary to limit the point of examination, by using the stopper in the stethoscope. By this means we can trace the precise boundaries of the pectoriloquy of a cavity; but when we try to find where the resonance of bronchophony ceases, we fail to find any exact limits; it gradually loses force as the tubes become smaller, or the superjacent lung more porous. Pectoriloquy is most characteristic when it forms a *little island of voice* under a clavicle, and little or no sound is transmitted nearer the sternum. The pectoriloquous bronchophony of a lung consolidated by inflammation, or compressed against the walls of the chest by a liquid effusion, never has this isolated character, but is generally louder in proportion to the size of the tubes involved in the condensation. It is however true that sometimes, in phthisis, the pectoriloquy is not thus circumscribed; for, besides the cavities, there may be general consolidation of the lung, and consequently free transmission of the voice over an extent of surface. Even in this case, a practised auscultator can distinguish the peculiar phenomena of cavities, in the snuffling, blowing, or tinkling and more articulate voice that certain spots present, or a coarser gurgling if liquid be present. It is easy to perceive that all these phenomena are liable to be interrupted or modified by the accumulation of the matter secreted by the cavities and adjoining tubes. So also as, in time, the disease advances, the excavations become extended, and the gurgling first, and the pectoriloquy afterwards, are heard in new spots.

It might be supposed that the excavation of tubercles, and the formation of cavities containing air, would remove the dulness on percussion that accompanies the two first stages of phthisis: but this is seldom the case; for although there is more air than there was, yet there is much solid deposit about the walls of the cavities, and the irregular density and flaccidity of the parts, as well as the deficiency of air in the peripheral structure of the lung, still tend to check and muffle the vibrations of the walls of the chest, and prevent them from yielding a clear sound. Even where the cavity is so large as to be the seat of a tinkling echo, or metallic tinkling, the resonance on percussion is irregular and imperfect; and thus may this case of metallic tinkling be distinguished from that of pneumothorax, in which some part of the chest must have an unnaturally clear sound. Sometimes the sound is clearer, in consequence of a general dilatation of the superficial cells; and as this is commonly of the flaccid kind, it may be accompanied by a sharp puerile kind of respiratory murmur: both these circumstances may disguise the phthisical signs, but only partially, for there will still be some decided irregularities in the sound on percussion, and enough of the signs of the subjacent caverns to declare the case to the wary observer. Now and then we find a case in which there is a hollow or bottle-like sound, on percussion over a cavity; this is when it is pretty large, and the intervening tissue is pretty uniformly condensed. More commonly there is an opposite condition; the walls of the cavity are loose and yielding, and, if it be large, percussion may sometimes cause a motion of its contents, and a gurgling or tinkling expulsion of air from it, which produces a muffled metallic sound, like that of money in the nearly closed hands, or more nearly like the imitation of that noise with which we amuse children in striking the hands, hollow and closed, upon the knee. Laennec compared the sound to that emitted by a cracked jar when it is struck.

With the irregular and deficient sound on percussion, generally most obvious under the clavicles or in other parts of the upper regions of the chest, there is very commonly associated a collapse or sinking in of the walls of the chest, forming under the clavicles a remarkable hollow, generally more conspicuous on one side than on the other. There is very commonly, also, some defect and irregularity in the movements of the chest; the upper part of one side being but little raised, and the lower parts altogether exhibiting the most motion. But there is seldom that complete fixing of one side that we see in chronic pleurisy, in which case, too, the upper part is generally more mobile than the lower.

We might class with the physical signs the characters of the sputa in the third stage of consumption, if they came only from the cavities which are peculiar to it. The expectoration of distinct portions of tubercle, or of pulmonary tissue, which have been seen in a few instances, constitutes a physical sign of the clearest character: they must come from cavities. If patients could save all their expectoration, and if this were inspected daily, this unequivocal sign might be more frequently met with. But the inflamed air-tubes are in great measure the source of the expectorated matter, which therefore presents much of the same aspect as in chronic bronchitis. The large size, and almost perfectly purulent character of the masses sometimes expectorated, which are like irregular balls of flock or wool, and of a yellowish or greenish colour, sinking and breaking down in water, go far to prove the existence of cavities in the lungs. A dirty yellowish brown or greenish matter, occasionally streaked or fringed with blood, flattening like a piece of money when separate, and in masses forming a smooth sluggish purilage, are more characteristic of phthisis, and commonly occur in the most advanced stage. The general pulmonary congestion which frequently precedes death, is often announced by the darker reddish or green hue of the purulent sputa. Profuse hæmoptysis does not happen often in the advanced stage of consumption; for the vessels soon become plugged with fibrin and obliterated in the diseased portions of lung, and the mass of blood is reduced to the capacity of those that remain free.

I think I have said enough of the most distinguishing signs of consumption to make it unnecessary to pursue them further into the details of differential diagnosis, or the distinctions between particular diseases. There is only one kind of lesion which, even in its physical signs, may be mistaken for tuberculous excavations; I mean dilated bronchi. These may be the seat of a coarse gurgling rhonchus, cavernous breathing, and pectoriloquy; and the accompanying chronic bronchitis often furnishes also a purulent expectoration. The situation, greater extent, and more stationary character of these lesions, may serve to distinguish them: they most commonly occupy the scapular, mammary, and lateral regions, and not the infraclavian; they usually extend over a considerable space, and they do not tend to spread as tubercular cavities do. Again, if they arise from disease in the bronchi only, they do not impair the sound on percussion so much as phthisis does: and if they originate in pleuro-pneumonia, the dulness is much more complete, and is confined to one side, and accompanied by a more marked contraction than that which occurs in phthisis. But we must take the general symptoms also into account. There is seldom, with dilated bronchi, the degree of emaciation that occurs in phthisis; and when they arise from condensation of the lung, there are very often œdema and general dropsy, which are not common in simple phthisis.

It is not at all uncommon for phthisis to become complicated with other diseases of the chest; especially bronchitis, pneumonia, and pleurisy, and the attacks of these additional lesions sometimes prove fatal even when the phthisical changes are not extensive. Thus it is very common to find general pneumonia attacking a lung in which there are miliary tubercles, which must have existed prior to the inflammation, and would probably not have run their course for many months. This complication greatly increases the danger of pneumonia, which, unless it be stopped in its very onset, commonly proves fatal. In some instances, especially in the young, we see a reason of its intractability in the tuberculous character of the hepatization, which has the grayish or boiled-liver aspect, with considerable softening, instead of the redder deposit of common hepatization; on the pleura there is sometimes seen, at the same time, the friable opaque lymph which borders so closely on real tuberculous matter: and in other instances, where the disease has not advanced far enough to present these appearances, we can still understand that there may be in the deposit enough of that defect of vitality which renders tuberculous matter so difficult of absorption. The supervention of the signs of an extensive pneumonia, crepitation, with increasing dulness on percussion, affecting the posterior lobes of one or both lungs, together with the increased febrile disturbance, increased heat, and the rusty tinge in the sputa, must be looked on as indicative of extreme danger to patients with any extent of phthisical disease; for if it do not itself prove fatal, as it very commonly does, the inflammatory attack will not fail to hasten and increase the phthisical disease. It is otherwise with the circumscribed pneumonia that not unfrequently attacks portions of the lung in the progress of tuberculous disease; these come on with much disturbance, and subside without causing much mischief, being probably the result of mere local obstruction or irritation. The same remark applies to the slight pleuritic attacks so common in phthisis, the effects of which are seen in the adhesions of the pleura so generally found in phthisical subjects. I have often heard a sound of friction in different parts of the chest, continuing for some days; then ceasing, and the chest, when examined long after, has exhibited adhesions at these points. I believe that the inequalities occasioned by the deposits in the lung may sometimes cause a local inflammation in the pleura, by their continual friction against it. Liquid effusions are less common, and arise from a more extensive cause, such as the bursting of a vomica into the pleura. If the vomica also communicate with the bronchi, there will be pneumothorax as well as liquid effusion. In either case the pleurisy is a serious and untractable addition to the consumptive disease, and may prove fatal in a few hours. Pulmonary hæmorrhage is another grave accident, occurring especially in the early stages of phthisis. It may prove fatal by loss of blood, or by suffocation; or the effusion of blood may break up the tissue of the lung to a great extent, and the patient may sink from the sloughy suppuration that ensues; or, after the hæmorrhage has ceased, inflammation may arise in and about the hæmorrhagic consolidation, and involve the lung in a destructive suppuration, which may be more or less of a tuberculous character. On all these accounts it is of the greatest consequence to guard against or to arrest pulmonary hæmorrhage, and when it has taken place, to use means to promote the absorption of blood from the tissue, and to prevent the rising of inflammation, which under these circumstances may prove unusually pernicious.

Destructive as phthisical lesions are, both by their own tendencies, and by the manner in which they affect the system, it is nevertheless now well ascertained that they do occasionally admit of cure. I think we are warranted in supposing that this may take place at any stage; but the mode of cure which has been most completely traced, is that by the expectoration of the tuberculous matter, and the lining of the cavity with a complete false membrane, which commonly is of a fibro-cellular or fibro-cartilaginous texture, tending to contract and ultimately to obliterate the cavity, but is sometimes thin and more like mucous membrane, without any obvious tendency to such a contraction. It is not uncommon to find in the lungs of those who have long laboured under symptoms of pulmonary consumption, some of the cavities with a lining more or less perfect, and at the apex of the lung especially there may be now and then found a cavity contracted almost to obliteration; and sometimes a mere cicatrix, perhaps enclosing a little friable caseous matter. All these instances evince a natural effort towards the healing of ulcerous cavities in the lungs; and where the disease is very limited in extent, and fresh tuberculous deposits do not take place in other parts, this healing of the cavities may amount to a cure of the consumption. The symptoms which may lead us to hope for such an unusual event, are a gradual diminution of the cough and purulent expectoration, a cessation of the fever, and quickness of pulse, and a decided improvement in flesh and strength. The signs that countenance this expectation are, the diminution of the pectoriloquy and cavernous respiration, and the restoration of some vesicular respiration, and a better sound on percussion in the part, whilst in the rest of the lungs the sounds are natural.

I cannot bring morbid anatomy to prove the possibility of a cure in the earlier stages of phthisis; but I have the history of several cases in which the signs render it extremely probable that some of the depositions which form the first stage of consumption of the lungs, had been removed by absorption. I have not space to give the detail of these cases: but their general features are these: there have been cough, some shortness of breath, in two instances hæmoptysis, in some pain under the clavicle, quickened pulse, some evening fever; and these symptoms, instead of taking the course of an ordinary cold, have remained permanent for two months and upwards, with some loss of flesh and strength. On physical examination there has been found some dulness on percussion, on or under one of the clavicles or scapular ridges, with somewhat bronchial respiration, or a slight permanent rhonchus, and increased resonance of the voice. Under treatment and favourable circumstances of air and climate, these symptoms and signs have been gradually removed, in some cases entirely, in others partially, but to such an extent that the patient's health has been considered in a great degree restored. It may perhaps be said that we have no certain proof that these cases were phthisical, or if they were so, that the phthisical lesions were entirely removed. I admit this, but maintain that the existence of phthisis was as much proved as it ever can be in its early stage,—that precisely similar cases when neglected commonly run a consumptive career,—and we are not aware that such a combination of signs and symptoms can be produced but by lesions which essentially have a phthisical tendency.

Malignant Diseases of the Lung.

The lungs are sometimes the seat of the malignant growths of encephaloid disease. It may form a separate tumour, but more usually the growth pervades the structure and consolidates it. The physical signs are then like those from hepatisation or tuberculation, from which the case can be distinguished only by the history and general symptoms, and by the absence of the constitutional indications of tubercles. These deposits commonly cause death, either by their encroachment on the function of the lungs, or from being simultaneously deposited in other organs, such as the mesentery, the liver, the ovaries, &c. But when they occupy the lung chiefly, both encephaloid and melanose deposits tend in time to soften and form ulcerous cavities, as in the case of tubercle. I have seen such cavities more than once in both these forms of disease.

The general appearance of *encephaloid disease*, or *medullary sarcoma*, is that of a brain-white solid, of varying consistence, with a pinker hue than that of tubercle, occurring either in separate tumours, which are sometimes encysted, or infiltrated through the tissues of the lung, and modified by their colours. When occurring in separate tumours, it is sometimes soft and cellular; in other cases tougher, and more like the pancreas; in others again, it becomes of fibro-cartilaginous hardness. A predominance of a loose cellular and vascular structure in it, with patches of extravasated blood, gives it occasionally the appearance that has obtained for it the name of fungus hæmatodes.

The only form of disease which I have seen affecting the lung, which approaches in any degree to *scirrhus*, is that which I have already described as a result of a chronic pleuro-pneumonia; there being in these cases firm adhesions to the pleura, a shrunk state of the lung, and dilatation of the bronchial tubes. The induration and glistening texture which the lung so changed sometimes exhibits, especially around the larger air-tubes, might lead one to suspect it to be of a scirrhous nature; but I have seen neither the tendency to cancerous ulceration, nor the simultaneous occurrence of scirrhus in other parts, which might be expected if this induration of the lung were really of a malignant kind.

I have met with several cases of *melanosis*, or *black tubercle*, affecting the lung, both exclusively, and with the same production in other parts of the system. In one case it was combined with encephaloid disease. The black matter may occur infiltrated in a natural structure, or in distinct tumours or deposits of an irregular cellular organization. I am much inclined to adopt the opinion of Andral, that the black matter is nothing but a modification of the colouring matter of the blood, in which carbon is in excess, or even in a free state. In the compound case just adverted to, the deposits exhibited in different parts various shades of colour, from the dark cruor red of hæmorrhagic engorgement to the deep jet black of perfect melanosis. The intermediate colours were of a bistre or sepia brown. The organized texture of melanose tubercles and tumours presents considerable variety, sometimes approaching to the most perfect products of acute inflammation, being soft and cellular or membranous; and sometimes having almost the totally unorganized structure of scrofulous tubercle. Probably it is only this latter form that undergoes the changes of softening and ulceration ascribed to melanosis by Laennec; and, under these circumstances, such changes are to be referred to the same causes as those which operate in the kindred changes of tubercle. The presence and modification of the colouring matter of the blood seem, therefore, to be the essential pathological condition of this disease, as an altered or deficient vitality of the fibrinous matter is of tuberculous affections.

It is necessary to beware of confounding with melanosis the accumulations of the black pulmonary matter, which take place to a great extent in the lungs of old people, especially among the inhabitants of large towns. These are probably, as Dr. Pearson supposed, derived from the soot inhaled with the air; which, I presume, finds access to the texture of the lungs chiefly through abrasions, softening, or other such lesions of the bronchial membrane, which, in a slight degree, often result from a common cold or cough. Whether from this source, or, as others have supposed, from an altered state of the colouring matter of the blood itself, I think it is plain enough, that when once deposited in any corners out of the immediate sweep of the circulation, such as in the angles of lobules, near old lesions, around large vessels, and in the bronchial glands, there it must lie, accumulating until death, or until it is carried off by the destruction of the tissue by some pulmonary disease. For it consists entirely of carbon; and this, being totally insoluble in any animal fluid, is scarcely susceptible of absorption, which cannot act on insoluble solid matter. For the same reason the carbonaceous matter of tattooed skins, and the insoluble oxide or chloride of silver in persons coloured blue from the too long internal use of nitrate of silver, are permanent, and can only be removed with the skin itself. It does not appear that this carbonaceous deposit in general interferes materially with the function of the lungs; but there are some curious cases on record, in which it has taken place so rapidly and extensively as to cause chronic inflammation and consolidation of a perfectly black colour, which tends to ulceration and the formation of cavities, as in other cases of chronic consolidations. Such cases are described by Drs. Gregory, W. Thomson, and others, as occurring particularly in coal-mines, and in persons labouring under bronchial disease whilst continually employed by the light of smoky lamps.

The general symptoms of encephaloid or melanotic consolidation of the lungs are those of obstructed breathing or circulation, dyspnœa, lividity, and dropsy; more commonly than those of consumption and emaciation, which belong rather to tuberculous disease. This is explained by their more rapid developement, and their not so readily leading to softening and ulcerative destruction of the organ. When this process does occur, the expectorated matter may afford means of distinction. I have seen, in the case of encephaloid disease, streaky red and white purilaginous liquid sputa, and, in melanosis, a considerable quantity of black matter, mixed with a muco-purulent compound. But such cases are not common, and the expectoration is more usually that of the bronchitis or pneumonia that may accompany the disease.

The *bronchial glands* are not unfrequently found after death in a diseased state, even when no symptoms referable to them had been manifested during life. I do not mean the deposit of black matter like that of the lungs, for that is so constant that it can scarcely be considered to be morbid. But they are sometimes found swollen and red, or containing caseous matter, or osseous concretions. In children they are occasionally so enlarged by the deposition of tuberculous matter, as to press on the air and blood vessels, and, according to Dr. Carswell, to produce dyspnœa and symptoms of obstructed circulation. They sometimes soften, and become evacuated by ulceration into the bronchi. Encephaloid disease, as well as extensive enlargement, not of a malignant character, may also affect these glands. I suspect that encephaloid disease of the lungs generally originates in this way, and spreads afterwards along the vessels into the pulmonary tissues. I have also met with cases of dulness on percussion on the top of the sternum, with signs of obstructed circulation and respiration, with simultaneous enlargement of the axillary and cervical glands, betokening a glandular tumour about the root of the lungs; and all these symptoms gradually subsided under the use of iodine and alkalies: so I conclude that the tumour was simple glandular enlargement. Considerable tumours of the bronchial glands might perhaps sometimes be discovered by dulness on percussion on the upper portion of the space between the clavicles, and on the spinous processes of the upper dorsal vertebræ. The tumours, generally, however, grow forwards, and I have seen them pushing out the sternum or the ribs on one side, and causing dulness at those parts, and symptoms of displacement of the lung further down. If they press on either branch of the trachea, they may cause a sonorous rhonchus, or more complete obstruction to the air on that side, with impaired motion and breath-sound, but unaltered stroke-sound. They also may produce signs by their pressure on the great vessels, arterial and venous.

PART III.

THE PATHOLOGY AND DIAGNOSIS OF DISEASES OF THE HEART AND GREAT VESSELS.

In investigating the signs of the diseases of the heart, the discoverer of mediate auscultation was less successful than with those of the lungs, and it is a proof of the soundness of his judgment that he candidly avowed his distrust of them. If some of his successors in this field of inquiry had shown the same candour and caution in their writings on the subject, the diagnosis of diseases of the heart would soon have been rendered as intelligible and sure as that of diseases of the organs of respiration.* Laennec did much for this subject, but his erroneous notions on the causes of the sounds of the heart vitiated many of his observations. These were partially corrected, chiefly by the investigations of the late Professor Turner, Dr. J. Hope, and Mr. Carlyle; but the true causes of the sounds were first satisfactorily demonstrated in the experiments which I conducted in February 1835, and which were detailed in the last edition of this work. These experiments have been repeated and varied by the London and Dublin committees of the British Association, as well as on the Continent and in America, with results generally similar. They have thrown much light on the pathology of diseases of the heart, which has consequently assumed a clearer and more intelligible character. There still remain some points of obscurity, and complicated cases of disease sometimes arise in which the signs are much disguised and modified by great changes in the condition of the parts.

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* Dr. Hope, in his work published in 1832, and republished in 1835, and in the Cyclopædia of Pract. Med. in 1834, gave rules of diagnosis for diseases of the heart, by which he stated, these diseases "may be detected with a facility and precision which can scarcely be surpassed in any other class of affections." In 1839 he expresses the same confidence, not in the same signs, but in others quite different, and in some instances opposed to the former. The *new* rules of diagnosis are, in principle, and in some cases in letter, the same as those published by me in the third edition of this work in 1835, and more fully explained in my lectures delivered in 1836 and 1837, and published in the Medical Gazette in 1838. Whether he derived his later views from my writings or from his own increased experience, I leave the reader to judge. I would only refer to the dates of our respective publications.

CHAPTER I.

PHYSICAL EXAMINATION OF THE HEART.

Position of the Heart.

A REFERENCE to the diagrams (see p. 3, 4, 5.) will remind the reader of the normal position of the heart, which should be familiar to the observer.

The heart is placed in the anterior mediastinum, rather to the left of the mesial line, and so oblique that the apex points forwards and downwards to the left, while the base lies back nearer the posterior centre, the spine. It therefore lies with its point on the diaphragm, underneath which are the liver and stomach; and it is bounded on other sides by the lung, except a small space of about two square inches, where, enveloped in its pericardial coverings, it is in contact with the walls of the chest at the lower third, and to the left of the sternum. The portion in contact is exclusively the right ventricle, but at each pulsation the apex of the left ventricle also strikes the space between the cartilages of the fifth and sixth left ribs, either through or under the thin margin of the lungs. The bulk of the heart and its great vessels lies covered by the lungs, behind the lower half of the sternum, and to its left from the upper edge of the third to the upper edge of the sixth rib.*

Manual Examination of the Heart.

On applying the hand to the left front of the chest, between the fourth and sixth ribs, the pulsations of the heart may be distinctly felt; but they vary remarkably both in strength and extent, according to the stage of the respiratory act and the position of the body, as well as from differences in their own strength. In a well-formed chest, the impulse of a healthy heart is scarcely if at all perceptible when the individual takes a full breath, or lies on his back. On making a full expiration, on the other hand, or on stooping forward, especially to the left side, it becomes strong, and is spread over a considerable surface, being felt much higher than usual. The position of the heart is not fixed in relation to the walls of the chest, but it hangs to a certain degree loose, and liable to displacement by change of posture and by the motions of the chest. The common position, however, is such, that only the apex is felt beating at a very circumscribed spot between the cartilages of the fifth and sixth ribs; in males, commonly about two inches below, and in front of, the left mammilla, and about the same distance from the left margin of the lower sternum. A deep inspiration elevates the ribs without raising the heart, which rather descends with the diaphragm; hence the apex then beats below the sixth rib; the impulse is, however, diffused, and scarcely perceptible, because the chest expands as the ribs rise, leaving the heart, and drawing the porous lung in front of it. (See the 3d fig. p. 5.) A forced expiration, on the other hand, depresses the ribs, and transfers the strongest pulsation to between the fourth and fifth ribs, and by bringing down the walls into contact with more of the heart, makes its impulse perceptible over an extended space, as high as the third rib, and on the lower half of the sternum. All these circumstances, although wholly unnoticed by Laennec and other writers, are of great importance, and should be known, not only to guard us against undue comparisons, but also to furnish us with the means of testing the free state of the heart and lungs, and of bringing the former more within reach of our examinations. Whilst we feel for the impulse of the heart, we should therefore desire the patient to vary his posture, by leaning forward on our hand, and first to take in and then to give out a full breath. The natural impulse will, for the same reasons, vary according to the form of the chest, and the extent to which the lungs are developed in front of the heart. In narrow or distorted chests, and in those contracted after pleurisy, the impulse of the heart may be felt much more extensively than usual. So also circumstances displacing the heart, such as tumours, and effusions of liquid or air into the pleura, may greatly change the character and degree of the impulse, diminishing or increasing it according to whether the displacement of the organ is from, or to, the walls of the chest. Abdominal tumours, and even a distended stomach, may, to a certain degree, have a similar effect. It must be remembered, also, that the impulse or perceptible motion of the heart is naturally intercepted or circumscribed by the soft porous tissue of the air-filled lung, and that it may be therefore modified by changes in that tissue. Hence pulmonary emphysema may still further intercept or circumscribe the impulse: consolidation of the lung, on the other hand, will propagate it over an extended space.

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* Dr. Hope is in error when he describes the aorta and pulmonary artery in contact with the sternum. Before the lungs collapse on opening the chest, they overlap these vessels. Hence the clear stroke-sound of the midsternum.

The greater thickness and strength of the left ventricle make its motions more forcible and extensive than those of the right; and it is, perhaps, for this reason that this ventricle is placed to act chiefly on the soft cushion of the lung, which offers no unpleasant resistance to it. We see by the position of the heart that the apex is almost the only part of the left ventricle that comes far enough forward to strike the walls of the chest. The front is constituted by the right ventricle, which lies under and even a little to the left of the sternum, and it is chiefly the impulse of this ventricle that is felt there on a full expiration, or on leaning forwards. It is by the comparative degree of impulse to be felt there, and further to the left, where the apex strikes, that Dr. Chambers chiefly judges of the relative condition of the two sides of the heart.

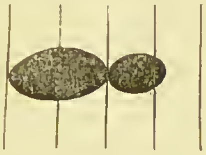
Auscultation of the Heart—Percussion.

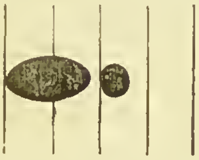
The examination of the region of the heart by *percussion* should be immediately added to that by feeling, and will be found to afford satisfactory illustrations of its results. In a well-constituted chest of a person who is not fat, there is commonly some dulness on percussion from the left margin of the sternum to the extent of between one and two square inches towards where the impulse is felt; but this dulness is remarkably diminished, if not quite removed, by a leaning back or supine posture, and by taking a full inspiration; and it is as notably increased by leaning forwards and to the left, and by a forced expiration. This is, then, another method of testing the free condition of the heart, and of the lungs that involve it. For example, if such variations are not observed—if in common states of respiration, and especially if on full inspiration, or on leaning back, there is a considerable extent of dulness on percussion at and to the left of the sternum—it may be concluded either that the heart, from adhesions or from its bulk, cannot recede from the walls of the chest, in which case the impulse would also be increased; or that there is a considerable effusion in the pericardium, in which case the impulse would be diminished.

To define the limits of the heart by percussion, we have to attend, not only to the *perfect dulness* which exists where the organ is in absolute contact with the walls of the chest, and which, in disease, sometimes extends over an area four or five inches in diameter; but we must notice also the *shading off* of this dulness, where the lung overlaps the organ. (See the diagrams.) To discover this we must use forcible mediate percussion, which gives the sound of the deeper-seated contents. By this means we may sometimes trace an enlarged heart when very little of it comes in contact with the walls of the chest. In emphysema of the lungs the permanently distended state of the anterior lobes nearly intercepts all impulse, and makes the region of the heart quite resonant; but strong percussion may still indicate that there is an enlarged dense organ beneath this resonant surface. In considerable enlargement, the sound of strong percussion on the left back, and in the left lateral region, is also somewhat impaired.

Auscultation of the Heart-sounds.

We now come to consider the examination of the *acting heart* by the sense of *hearing*. I shall first describe the chief facts of the subject, and afterwards enter into an explanation of those facts. If we listen either with the stethoscope or with the ear alone applied to the region of the heart, we hear at each pulse two sounds following each other in quick but regular succession, and succeeded by an interval of silence until the next pulsation. The first is a long, rather dull sound; the second is a short abrupt flap. Some French writers have used a very wrong sounding word for this double sound, by calling it a *tic-tac.* If it is to be given at all by the symbols of articulate sounds, the word *lubb-dŭp* will best express it. Laennec endeavoured to give expression to these sounds, by marking their rhythm or measure. Supposing the period of a pulse (that is, the time from the commencement of one double sound to the commencement of another) to be divided into four equal parts, he described two of these as occupied by the first sound, a little more than one by the second sound, and the remaining less than one by the interval of silence. These sounds present much variety, so that it is difficult to estimate their proper length; but I should note the average rhythm somewhat differently from that given by Laennec, who makes the first and second sounds longer, and the interval shorter, than what I have generally heard. I will represent the measures by a diagram, which will be more generally intelligible than musical notation. The first is that given by Laennec; the other is what appears to me the more usual rhythm in health.





This is the general character of the sounds in the healthy male adult. In females and in children the first sound is rather shorter and less dull, more like the second; and the same difference may be observed when the pulsations of the heart are frequent or weak.[*]

Causes of the Sounds of the Heart.

The physical causes of these natural sounds of the heart have been the subject of much discussion. Laennec's opinion was, that the first sound was caused by the contraction of the ventricles, and the second by the contraction of the auricles;

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[*] The period and duration of the sounds of the heart are further given in table iii. at the end of the work.

but he did not advance any proofs in support of this opinion, nor did he attempt to explain how these respective contractions could generate the sounds. The late Professor Turner of Edinburgh was the first to test the matter by physiological experiment; and he was thus led at once to detect the error of Laennec, by observing (what had been before noticed by Harvey and Haller) that the contraction of the auricles immediately precedes that of the ventricles, and therefore cannot cause the second sound. This observation of Professor Turner was afterwards confirmed by the experiments of Dr. Hope, in which it was proved that the first sound distinctly accompanies the contraction of the ventricles, and that the second sound occurs at the moment of their diastole, and is in no way connected with the motions of the auricles, which appear to be generally too slight to cause any sound. But the immediate physical causes of both sounds were undetermined until February 1835, when, assisted by Dr. Hope and others, I conducted a course of experiments which led to pretty conclusive and satisfactory results. These experiments were afterwards repeated and varied by the Dublin Committee of the British Association for the Advancement of Science; and more recently an extended series of experimental researches on the same subject has been conducted by Drs. Clendinning, Todd, and myself, in another committee for the same association. The different reports of these investigations will be found in the Transactions of the Association, and in the Medical Gazette. It is satisfactory to me to be able to say, that they confirm, in all material points, the results and conclusions of my first experiments, first published in the third edition of this work. These experiments, together with various questions and opinions which they affect, I reserve for an appendix, rather than confuse the student by introducing them in the text. I will here introduce such chief facts and conclusions as may enable us to comprehend the true nature of the sounds of the heart.

It was decided unequivocally, by all the series of experiments alluded to, that the first sound accompanies the whole duration of the systole of the ventricles, which also causes the impulse felt on the walls of the chest; it was equally clear that the second, or flapping sound, occurs at the first moment of the diastole, and that the motion of the auricles was not accompanied by any perceptible sound.

Then, first, how does the contraction of the ventricles produce the first sound? Sound we formerly defined to be *motion of a certain force resisted with a certain force:* where is the resisted motion in the contraction of the ventricles? One of the first ideas that suggest themselves is that of Magendie: does not the heart produce the impulse by striking against the walls of the chest? and why should this not cause sound? I answer, that in forcible pulsations, and when the lung does not too much intervene, I have no doubt that the impulse does produce sound; and if we listen to the sound of the heart when it is beating strongly, or when, by leaning forward or by breathing out, the heart is brought in contact with the walls of the chest, we hear the first sound has something like a knock in it, which we can can scarcely help referring to the impulse. But this is an accessory, and not an essential sound; for we may hear the first sound when there is no impulse, as in a person leaning backwards, or taking a full breath; and in our experiments we heard the first sound at the origin of the arteries when the body of the heart was surrounded with the soft lung or with tow, or was allowed to hang out of the chest, and strike against nothing in its motions. It is well known, too, that liquid in the pericardium, or liquid or air in the pleura, although it entirely prevent the heart from striking the walls of the chest, does not annul the first sound. In moderate pulsations the heart makes a partial rotatory movement; the long fibres of its anterior convex surface drawing the apex upwards and forwards, and causing it to slide obliqnely on the smooth pericardium, bring it to the walls of the chest too gently to produce sound, except under the circumstances before mentioned.

The first sound, then, is produced by something in the heart itself, either by its contents or by its own structure. Can it be by its contents? by the motion of the blood resisted by the inequalities of the interior of the ventricles? This was supposed by the Dublin Committee to be the chief cause of the sound. But the motion of the blood over these inequalities is not rapid, nor is their resistance considerable at the time of the production of the sound. These inequalities are, as Dr. Cowan has pointed out, chiefly confined to the lower or auricular portions of the ventricle, into which the blood has already passed before the systole begins; and the effect of the systole is to drive the blood, not over or across the inequalities, but from them, to those smooth and funnel-shaped parts of the ventricle which lead to the arteries, and which offer the least possible resistance to its course. The contraction of the ventricles propels the passive mass of blood gradually, that is, during the whole period of the first sound, into the arteries; and the motion is therefore too slow and easy to be likely to cause sound. Some of our experiments further settled this point, by proving that the first sound continued when the ventricles contracted without any blood in them.

By excluding the blood we are thus brought to the conclusion that the cause of the sound must be in the solid structure of the ventricles: it is our next question, whether it be in any part of them in particular. Several writers have ascribed it to the auriculo-ventricular valves, which, when they close, are supposed to produce a flapping sound. But the act of closing these valves is momentary, and takes place only at the commencement of the systole; whereas the first sound of the heart is prolonged through its whole duration. Further, in some of our experiments the first sound continued, although impaired, when the auriculo-ventricular valves were prevented from acting, by fingers introduced into their orifices, or by some of their cords being *hooked* back or cut. Still these valves no doubt produce a part of the sound, for at each contraction they are suddenly tightened in a manner calculated to generate sound.

But are the valves the only parts which are tightened at each systole of the ventricles? Is not every muscular fibre in the ventricles suddenly tightened by this action? Here are the elements of sound, motion vigorous and rapid, suddenly resisted by the mass of blood to be urged forwards by the contraction, and the contracting motion and the resistance, although greatest at first, continue to act as vibrating forces during the whole systole; hence the prolongation of the sound. In other instances, abrupt and forcible muscular contraction produces a sound like the first sound of the heart; as when we apply the stethoscope to the adductor muscle of the thumb of the closed hand, and contract the muscle strongly and quickly. Or, to avoid the possibility of the joints being the seat of the sound, if we apply the end of a flexible tube to the abdominal muscles, and start them into sudden vigorous action, we may thus get sounds quite as loud as those of the ventricles, and very like them in character.

By varying the mode of this muscular action, different kinds of sound may be produced. When the contraction is slow or sustained, however strong, we have only the dull rumbling noise which Dr. Wollaston first described, and which he attributed to a vibration depending on a regular intermittence in the force of the contraction. When the contraction is gentle and slow, it may cause no sound at all; as we have seen that the auricles generally produce no sound, neither do the ventricles, when their contraction is very feeble. But whenever there is strong abrupt muscular action in any part of the body, like that of the heart, there will be heard a sound which will resemble that of the ventricular systole, in proportion as the muscles in which it is produced resemble in thickness and density the tissue of the heart. The loudness of the sound is by no means in proportion to the thickness or strength of the muscle, but rather to its simplicity, and the abruptness as well as the vigour of its contractions: the transition of a thick muscle from slack to tight can never be so complete and sudden as that of a thin one; where there are many fibres, they choke or muffle each other's vibrations; hence the sound is dull and prolonged, rather than loud and clear. Many writers who have objected to my explanation of the first sound of the heart, have done so in ignorance of the principles on which muscular action causes sound; when these are known, the identity of the phenomena becomes apparent; and in my experiments there was the best proof that we could have, that the muscular contraction of the heart produced systolic sound, for we had the heart out of the body, without its blood, without valvular action, lying on the table, or on my hand, and its contractions were still accompanied with a sound, weak indeed, but in character resembling its natural first sound.

The walls of the ventricles appear to be peculiarly calculated to generate sound; their flaccid state when relaxed, the fineness of their fibres, and the harmony with which they suddenly contract on their contents, and become almost as hard as a stone, (as we can feel in the living heart of a stunned animal,) fulfil the conditions best calculated for the production of sound. The commencement of the systole, producing the tightening of the auricular valves, and thereby completing the resistance of the body of blood on which the contracting fibres have to act, is naturally its loudest part, and often has a flapping character; that which continues after is more dull, and is prolonged according to the quantity of blood to be expelled, and the continued strength of the contraction. This prolonged termination of the sound is, therefore, best heard when the heart acts slowly and vigorously.

And now what causes the second sound? That it is intrinsic, and not caused, as Magendie supposed, by the heart striking any of the surrounding parts, we proved by the same experiments, in which the intrinsic character of the first sound was shown: the sound continued when the heart was so completely isolated that it could strike against nothing. What is there, then, within the heart, that can produce this short clapping sound at the moment of the diastole? Is there any thing that tightens at that moment? Not the walls of the ventricles certainly, nor the auricular valves, for they are then all loose and flaccid. What can it be but the semilunar valves at the mouths of the arteries, which are then suddenly tightened by the reaction of the arterial column of blood? And so it was proved to be, in my experiments first, and in many repeated since; for by hooking back these valves, or by pressure preventing the reaction of the column of blood upon them, the sound was stopped; and by releasing the valves, or discontinuing the pressure, the sound was as surely restored.*

Considering it, then, established that the first sound of the heart is produced essentially by the tightening of the valves and walls of the ventricles by muscular contraction, and that the second sound is caused by the sudden tension of the arterial valves by the recoil of the arterial column of blood upon them, we proceed to examine how far these sounds may represent the condition of the parts which respectively produce them; how they may be modified by changes in these parts, and by the addition of new elements of sound arising from such changes.

The elements of the first sound are the contraction of the walls of the ventricles, and the resistance given to their contraction by the blood within them. Each of these elements may vary in character, and give corresponding varieties in the nature of the sound. Force and briskness in the contraction must together increase the sound; briskness gives it loudness, but not duration, which is rather caused by strength and continuity of action. Hence a heart that is contracting abruptly may generate a loud sound, even when its contractions are not strong; and a heart acting strongly, but slowly, will produce a duller sound, of longer continuance. These different modes of contraction are frequently dependent on the thickness of the walls of the ventricles; but not constantly. We can readily understand why the contraction of a thin muscle should be abrupt and short, from its simplicity and want of mass; and why a thick muscle should be slower, although stronger in motion. If we observe the different sounds produced on tightening thin silk and thick baize or cloth, we find that the thinness of the silk gives a unity and briefness to the impulse which it receives, and the sound is short and loud; whilst in the baize the impulse is divided and prolonged in the complexity of the fibres, and the sound is dull and less brief. So a thin ventricle will give a louder, sharper sound than a thick one, under similar circumstances. But when, under the influence of increased irritability, a thick ventricle contracts with more abruptness than strength, it may give as short and as loud a sound as a thin one which is acting slowly and with more tone. I have frequently heard a hypertrophied heart, when excited by nervous irritation, produce sounds louder than those of a dilated heart that was acting quietly. In this case the stronger impulse from the thicker heart would still distinguish it; but there are some conditions in which even this test may fail: for instance, during or after syncope, the action of the heart is abrupt and feeble; and even when the walls are thick, the sound is generally short or flapping, and the impulse weak. It would seem that the systolic action is incomplete or partial, confined perhaps to the outer fibres and the tension of the valves; being abrupt enough to be sonorous, but insufficient to cause an impulse. These variations, which are exceptions to the rules given by Laennec and other writers, are explained by the view which we take of the production of the sound, and ought to be known, or they may lead to error.

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* Dr. Carswell seems to have been the first who conceived that the second sound is caused by the flapping of the semilunar valves; he was led to this opinion by observing the signs of a case of aortic aneurism in 1829, a drawing of which is now in the museum of University College. The first proposal of this explanation that I know of in print, is in a thesis by Dr. Elliott, (De Corde,) Edinburgh, 1831. Dr. Billing, M. Rouanet, and Mr. Carlile, were later advocates of this view.

Although the ventricles are two, yet their fibres are in great part continuous, and their action and sound is one. But the different thickness of the two causes the sound to be shorter and clearer at the sternum which is over the right ventricle, and duller and longer where the apex of the left ventricle beats between the fifth and sixth ribs; and these differences are more remarkable where, in consequence of disease, the difference in thickness is augmented.

The other element contributing to the production of the first sound is the resistance opposed to the contracting ventricles by the mass of blood within them. Now the amount of this resistance will depend on the quantity of the blood, and the opposition that it meets with in its passage from the ventricles. When the quantity of blood is great, it will resist the ventricular contraction longer, and therefore maintain the sonorous tension longer than when it is small; but the resistance will not be so sudden, and the sound not so loud: for if the heart be distended with blood before the systole begins, its fibres cannot be so loose, and the great cause of sound, the transition from slack to tight, will be less complete. On the other hand, when there is little blood in the ventricles, they pass from great flaccidity to tension so suddenly that the sound may be unusually loud, but it can have no continuance. As far as the blood is concerned, therefore, a dull prolonged sound is a sign of a large quantity, and a short loud one of a small quantity, expelled from the ventricle. In accordance with this, we find that when the pulse is slow, other things being equal, the sound is much longer than when it is quick; and such a length of sound in a pulse that is not slow, and the heart not diseased, becomes a sign of sthenic plethora. The longest first sound that I ever heard was in a gentleman whose pulse beat only twenty-eight in a minute; it occupied nearly a second of time. But this is not the case with all slow pulses, for where the pulse is low from weakness, as in syncope, its contractions may be brief and incomplete, and the sounds short and weak. A short sound with a slow pulse is essentially a sign of weakness, and this may occur in asthenic plethora or cardiac congestion. When there is little blood in the system, we find the sound remarkably short and flapping, and it may be very loud; but then the pulsations are commonly frequent, and the radial pulse small.

But besides the quantity of blood, the resistance to its expulsion from the ventricle may modify the character of the first sound. If the vent for the blood be free, the resistance will be brief, and the sound short. Now the chief resistance opposed to the blood is that of the semilunar valves tightened by the pressure of the arterial column, and this will be great in proportion to the fulness of this column. Here is then another reason why an abundance of blood tends to lengthen the systolic sound; there is more blood to be expelled, and more resistance to its expulsion. On the other hand, when there is little blood in the arteries, the systole takes place suddenly, with little resistance, with a short sound, and throws the blood into them in a loose sudden jerk, which is often felt in the pulse of those who have lost much blood. A permanent narrowing of the arterial orifice by disease must also tend to lengthen the first sound by prolonging the systole; but as such a change developes a new sound or murmur at the constricted point, it will be better to notice it afterwards. There is, however, one more element in the resistance given to the systolic contraction which must be noticed as a chief cause of the commencement of the sound; that is, the closure of the auriculo-ventricular valve. Until this takes place, the resistance scarcely begins, for the passage back into the auricle is too free; but no sooner have these valves closed, than the ventricles tighten on the ball of blood, and the sound is produced. We can see then how the free perfect play of these valves contributes to the first sound, not only by their own tightening, but also by their completing the strain of the other walls of the ventricles. The closure takes place suddenly, almost at the beginning of the systole, and thus causes the loud and flapping commencement of the sound. Where from disease their closure is not sudden or complete, this flapping commencement is less perceptible. There may besides be a new sound or murmur produced by the current of blood through the unclosed valve, but this we shall consider by-and-bye.

I have not done yet with the varieties of the first sound; for, besides those of duration and loudness, there are other differences in its character.

Sometimes the systolic sound, without being longer than usual, seems broken into two by something like a flap in the middle; or to enunciate it, instead of being *lubb-dŭp* as usual, the first and second sounds are *bullub-dŭp*. I think it probable that this variety may depend on some irregularity in the action of the auricular valves, especially the mitral, by which their tightening with its attending flap is either delayed or takes place in two successive jerks. This kind of sound occurs most commonly in cases of diseased heart; but I have heard it sometimes where there was no reason to suspect permanent lesion, but where the action of the heart was languid. In these cases the impulse likewise is sometimes double, and from the same cause. A double impulse is also now and then produced by the action of the valves preceding or following with a jerk the stroke of the apex against the ribs; this, too, may make the first sound double.*

In some recent experiments with Dr. Clendinning, I found the auricles of an ass produce a very distinct sound when they contracted vigorously, and independently of the ventricles. This was afterwards heard by all who were present. The same phenomenon has been also observed in some experiments recently performed in America. It is possible, then, that the auricular contraction may sometimes cause the first sound to be double.

The systolic sound has sometimes a remarkable drum-like character, without any murmur or other irregularity, the double sound of the heart being then *lumb-dŭp* or *lung-dŭp*. As far as I can remember, this sound has been observed only in cases where there were signs of liquid in the pericardium or left pleura; and if this be the case, we may explain its more musical character by the contractions of the ventricles taking place in a medium which leaves their vibrations more free than usual, not muffled by a closely-wrapped membrane and the spongy lung. In these cases the impulse is diminished, and this separates them from those with the metallic clink which frequently accompanies the sounds of a strongly-acting heart, and which Laennec referred to air in the pericardium, whilst Dr. Hope and some recent French writers have imagined other causes equally fanciful. This metallic clink originates in the ear of the observer, and has nothing to do with the heart further than that it is excited by the blow which the heart communicates to the ear. We may hear the very same note by laying the ear flat upon the table, and tapping the under side of the table just opposite to it. I believe this to be the proper note of the tympanum. We often hear it in auscultation, but when once aware of it, it is easily recognised as belonging to the observer, and not to the patient. It is easily produced when the heart strikes against the ribs, and the external knocking sound is added to the intrinsic systolic sound.

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* This explanation of the double first sound was published in my lectures in the Medical Gazette in 1838. In 1839, Dr. Hope announced as *a discovery of his own*, that the double first sound is caused by the apex of the heart striking the fifth rib!

The addition of the knocking to the first sound is very perceptible when the heart beats strongly, or in the circumstances of posture, state of the respiration, and shape of the chest, before mentioned as most favourable to increase the impulsion of the heart against the ribs ; but it is so much incorporated with the intrinsic sound that it seems to form a part of it. This is not surprising when we consider that the sounds produced by at the same time striking and straining a body, must partake of the same character, and become incorporated, seeing that its tension and mode of vibration are for the moment uniform. The sound is no doubt partly generated also in the walls of the chest; and when the impulse is very abrupt, it gives the impression of a knocking sound.

Besides the intrinsic sound and that of impulse which is thus occasionally added to it, the heart's motions sometimes produce sounds in the adjoining parts. The friction-sound of the pericardium I shall notice hereafter ; but beyond this, a strong impulse of the heart on a portion of lung may forcibly press the air from it; and if there happen to be any partial obstruction or mucus in its tubes, a short sibilant or mucous rhonchus may accompany each beat. The character of these additions, and the circumstance that they accompany the breathing also, and are more or less diminished by holding the breath, or by posture altering the manner in which the heart beats on the lung, may serve to distinguish them from the true cardiac sounds. Again, I have repeatedly heard the tinkling echo of pneumo-thorax—of a large cavern—of dilated bronchi—nay, even of an inflated stomach, accompany the pulsations of the heart when excited; these circumstances may be easily detected by their other signs, as displayed by auscultation and percussion.

The *second sound* of the heart is more simple in its character and causes, and less liable than the first sound to be modified by circumstances. Caused solely by the sudden reaction of the arterial columns of blood on the semilunar valves, its loudness will depend on the mobile and perfect state of these valves, and the extent and abruptness with which they are stretched by the recoil of the blood at the moment of the ventricular diastole. It will, therefore, be most perfect when the heart acts regularly and slowly, giving time for a full gush of blood to carry the valves loose into their slight recesses in the walls of the artery, and for the as perfect reaction of the contents of the distended artery on their concave surfaces. On the other hand, if, from the quickness or weakness of the ventricular contractions, the jets of blood thrown into the arteries be small, the play of the valves will be less complete, and the sound less distinct. Also, if the permanent distension of the arteries be great in proportion to the propulsive power of the heart, as it sometimes happens during fits of palpitation, the valves may not in many pulsations be opened enough to flap distinctly. Or if the arterial tension be greatly diminished, as under the immediate influence of profuse hæmorrhage, it may not close them with sufficient force to give a sound. The second sound is generally heard most plainly at about the middle of the sternum, or between the third and fourth ribs close to it, which is opposite to the situation of the arterial valves; but it is generally audible over most of the upper parts and front of the chest; in the normal state perhaps more extensively than the first sound. Although there are two sets of valves, aortic and pulmonary, yet being brought into play at the moment of the diastole, which is simultaneous in both ventricles, they are generally tightened at the same instant, and produce but one sound. But if from the less mobile state of one set of valves, or from the greater pressure of the blood on them in one artery, the tightening of the two sets do not coincide, the second sound may be double, consisting of two flaps in quick succession, so that the note of the heart is then *lubb-dŭpŭp*, or *lubb-dŭrrŭp*. This sound sometimes closely resembles in rhythm the footsteps of a cantering horse. It rarely occurs (never, I believe, for a continuance) in cases where the heart is free from disease. When the action of the mitral valve is also retarded in the first sound, both sounds may have this double character.

It requires some practice in auscultation to enable the student to distinguish some of these varieties of sound; and if the action of the heart be irregular or quick, it is not always easy to distinguish between the first and second sounds. When the impulse against the ribs is pretty distinct, this being felt either by the ear, or by the finger applied at the same time as the ear, will sufficiently declare its companion the first sound. When the impulse is obscure, the finger may be applied to the pulse in the carotids, which is not perceptibly after it. The radial pulse is so much later, that it may not unfrequently even accompany the second sound. The different characters of the sounds will often serve to distinguish them; and when these are little marked in the region of the heart, they are often quite obvious at the top of the sternum, or in the carotid arteries, where the flap of the second sound can seldom be mistaken. The sounds in these arteries are, under ordinary circumstances, merely those propagated from the heart; but, under the influence of violent action, I have no doubt that a sound is produced by the shock of the blood against the sides of the great arteries, and is heard over them as a loud short sound, as intense as the simultaneous first sound of the heart, but more abrupt. So much for the natural sounds and the variations from them, all of which are seated in the solids of the heart and its appendages.

Morbid Sounds, or Murmurs of the Heart and Arteries.

We have argued by reasoning, and have shown by many experiments, (see the investigations before quoted,) that although sounds accompany the motions of the solids, yet the motion of the blood through the heart and vessels is habitually accomplished with the smallest possible resistance, and therefore wants one element essential to generate sound. But under various circumstances of disease this element, resistance, may be produced in the obstructions to the current of the blood, or in the novel channels into which it is thrown; and thus are developed new sounds, which, as distinct from the natural sounds, we may, with Dr. Forbes, class under the general term, *murmurs*. An example or two will set before us something of the character and causes of these murmurs.

At the moment when the blood is issuing from the ventricle, if, instead of a smooth, equally tense channel, it meets with a rigid constriction, or an abrupt orifice, its passage through it will be attended with a whizzing or blowing noise, which may be heard in the region of the heart. Or to take a simple case: if we apply the stethoscope in the course of an artery far from the heart, we hear nothing as long as the current of blood flows smoothly and unmodified through it; but if by pressure we diminish the caliber of the tube at any point, we then complete the elements of sound; we give resistance to the moving blood, and at each pulse we hear a blowing or a whizzing sound, which will vary in tone and loudness according to the force of the current and the degree of resistance which it meets with. This is purely a physical phenomenon; it may be produced in any tube through which a current of water runs. Thus, if we take an india-rubber tube, and adapt it to a reservoir of water, so that the water shall flow freely through the tube, we may, by pressure on the tube, produce murmurs, varying according to the force of the current and the resistance which the pressure opposes to it. They are sometimes like blowing; sometimes like rasping or sawing a piece of wood; and now and then they may be heard in quite a musical tone, which implies that the vibrations are then regular and sustained. The blowing and musical murmurs are generally caused by greater regularity, but less force of current, than that which produces the sawing or whizzing sounds.*

In fact, all these murmurs are produced by the passage of *liquids* through solid tubes or apertures, in the same manner as analogous sounds are produced by the passage of *air* through pipes or holes of different kinds. They are the music of *water-instruments*, as the latter are the music of wind-instruments. There are only these differences between them, that liquids, being more sluggish than air, are less susceptible of the sudden motions which constitute sonorous vibration; and not differing so much in density from the solids in which they move, liquids will have little of those reflected or echoed vibrations which increase and modify the sounds produced in air-filled tubes. Holding in mind these qualifications, we may explain the murmurs heard in the heart and arteries, by referring to parallel instances of the tones of wind-instruments; nay, we may find the parallel phenomena in the rhonchi, respiratory, and vocal sounds of that most complete and diversified wind-instrument, the windpipe and its branches. Like in these, there are varieties of sound, in generating which the solids and the current have different shares: thus in the grating, sawing, and stronger droning murmurs, the vibrating resistance of the solid is chiefly concerned; and its vibrations are transmitted to the adjoining parts as well as to the current, so as to produce in them a thrill which may sometimes be felt by the hand. Being in the rhythm of the heart's motion, this thrill resembles that felt on the back of a purring cat, whence Laennec called it the "frémissement cataire." These have their parallels in the sonorous rhonchus, in reed instruments, and in those imitations of these murmurs which we can produce by forcibly breathing through the nearly closed teeth, tongue, and lips, which in like manner communicate a sensible vibration to the solids, as in ringing the letter *R*, in a whisper. Again, in the blowing, hissing, whistling, and cooing murmurs, the vibrations are more those of the current reflected by the solid, in the manner of cavernous breathing, the sibilant rhonchus, blowing, or whistling with the mouth, or of the flute class of musical instruments. Here there are no perceptible vibrations in the solids; they are less actively concerned in the production of these sounds, which are rather transmitted in the direction of the current.

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* See the 2nd Report of our Committee in the Appendix.

Although I adhere to this view (the idea of which I first proposed in 1828) as the only one which gives a complete physical explanation of all cardiac and vascular murmurs, I must not omit to state that many other explanations have been proposed. Most of these are too vaguely stated to be really explanatory, being mere references to the indefinite influence of friction or obstruction. There is one, however, which is more precise, and none is more plausible or ably supported; it is that of Dr. Corrigan of Dublin, to whom we owe many interesting observations on the pathology and diagnosis of diseases of the heart and arteries.

Dr. Corrigan considers that murmurs are produced, not in the constricted or resisted portion of the current, but in the flaccid walls beyond, which are thrown into vibration by the rippling motion which the current assumes there. This view, he thinks, explains the occurrence of murmurs in the uterine arteries and in varicose aneurism, where, instead of constriction, there is an unusually free passage for the current; but, from the changed caliber of the vessels, it is of a rippling kind. Dr. C. has illustrated this view by a number of ingenious experiments, for an account of which I must refer to his paper in the Dublin Medical Journal. I think, however, that this view of Dr. Corrigan is partial, and mistakes an effect for a cause. The flaccid state of the tube beyond a constricted point is an effect of the impediment which the constriction opposes to the current, and the rippling motion of the current equally originates in the constriction, although, being once produced, it continues beyond it in the looser part of the tube. To prove this, if we cut off this flaccid part, and let the tube terminate with the constricted point, we still have the murmur produced in it. I have tried this experiment with a current of water through an elastic tube, and with an india-rubber bottle, from which, by sudden compression, there can be produced a current like that from the heart, and I have always found, that if I oppose a resistance to the current, either by pressing on the tube or mouth of the bottle, or by holding something in it, the murmur is produced as well at the terminal orifice as at any other point. In these cases the constricted portion is like the mouth-piece of a wind-instrument, it alone produces the sound; but when so produced, the tube beyond may receive and modify it. I have produced a murmur also with the systole of an ass's heart, after the arteries have been divided, by compression on their cut ends.*

How, then, are we to explain the murmur in the uterine arteries and in varicose aneurism, in which there seems to be no constriction? I would refer to the parallel case of a wind-instrument, the flute, in which also there is no constriction; for, after the wind has left the lips, there is free vent enough for it, and yet the sound may be of the loudest kind. There may be a vibrating resistance to the current without constriction: for instance, when the current enters a new and enlarged channel, at a considerable angle, and strikes against its sides, it communicates to them an impulse which they resist, and the series of impulses and resistances which thus ensue, if forcible and rapid enough, constitutes sonorous vibrations. The dilated and tortuous state of the arteries of the gravid uterus, and the unusual direction of the arterial current into a lateral or a varicose aneurism, present conditions well calculated to produce sounds in this manner.

I will now briefly state the chief pathological conditions causing different kinds of murmurs, which may prove signs of those conditions:—

1. Constriction or projection in the arterial orifices; causing a murmur with the first sound, blowing, rasping, or whizzing: very common on the left side of the heart, more rare on the right.

2. Constriction or impediment in the auriculo-ventricular orifices, causing a murmur with the second sound: very rare.

3. Imperfect closure of the arterial valves, causing a murmur from regurgitation, with or instead of the second sound, generally whiffing or grating, sometimes musical: very common in the aortic valves.

4. Imperfect closure of the auriculo-ventricular valves, causing a murmur from regurgitation, with, and often

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* These facts, so subversive of his theory, Dr. Corrigan has endeavoured to evade, by assuming that the sounds thus produced are not like the true bellows murmur; but if he will take the trouble to repeat the experiment, he would find them to be quite identical.

impairing, the first sound; generally blowing, sometimes whistling or cooing: very common on the left side.

5. Considerable dilatation or relaxation of the aorta or pulmonary artery, just above its orifice, which, although of normal dimensions, is narrow, compared to the tube beyond it; causing a murmur with the first sound, generally grating: rather rare.

6. Partial obstruction or projection in a blood-vessel; causing a murmur with the pulse; blowing, musical, whizzing, or grating; prolonged in proportion to the degree of obstruction: common.

7. Abrupt aneurismal dilatation of an artery; causing a murmur with the pulse; generally grating: rather common.

8. Unnatural opening from a ventricle, or from an artery into another cavity, sac, or vessel, causing a murmur with the pulse, generally whizzing or rasping; if from the ventricle, it must terminate with the second sound; if from the artery, it may continue beyond it: rare.

It may be generally said of all these murmurs that they are increased by augmented force of the heart's action, and they may sometimes be changed by this influence; the blowing becoming whizzing or rasping, the musical murmur raised in pitch, and the like. So also the constriction of an orifice or artery, the defect of a valve, or any of the other causes, may be present in so low a degree as to produce sound only under the temporary influence of an excited heart. On the other hand, I have met with instances, but they are very rare, in which increased action has diminished or even removed a murmur. The quantity of blood in either extreme may also modify these sounds: when excessive, it increases and prolongs them; when very defective, and accompanied by an excited action of the heart, it may make them loud and short. Even the quality of the blood may influence the sounds; for a thin watery fluid is more readily thrown into sonorous vibrations than one of a richer, more viscid character; and this is one reason why murmurs are so easily produced in chlorotic and anemic subjects. In these the slightest pressure of the stethoscope on the arteries and veins in the neck, is enough to cause a loud continuous or remittent whirring, to which the French have given the name of "bruit de diable," from its resemblance to the noise of a toy called a "diable."

It is in similar conditions that a murmur strongly generated at the orifice, or in the course of an artery, may be propagated through a great extent of the tube beyond, being carried with the current, which retains the vibrations, and may even communicate them to the finger by a sensible thrill, constituting what Laennec called "frémissement cataire." Sometimes the thrill is perceptible without the murmur; this takes place when the vibrations are too slow to produce sound. It gives the feeling of something rough passing rapidly under the finger, and is quite distinct from the hard throbbing pulse of arteries leading to an inflamed part.

I do not believe that either increased or diminished action is capable of producing a murmur in healthily constituted hearts and arteries. There must be something to modify, as well as to hasten the current, before it can produce sound. Neither do I see it possible that an altered shape of the ventricle can cause a murmur in the arterial orifice, without either constriction or projection in that orifice; or, what is equivalent to it, dilatation or relaxation of the artery beyond it. In fevers, inflammations, and nervous irritations, we have every degree of increased action; but no murmur, without other disease; and I have met with hearts enlarged and dilated in various ways, but producing no murmur, without some change in the orifices or arteries. This statement is not in accordance with the opinion of other writers; but it is given as the result of much experience and study, and it increases the value of murmurs as diagnostic signs, if by their characters or positions they can be traced to their mechanical causes.*

Arterial Pulse.

The *arterial pulse* is another sign by which we judge of the action of the heart. It is seldom so correct an index of the action of the heart as the sounds and impulse of the organ itself; yet some of those impracticable sages, who find physical signs in general too troublesome to be studied or valued, have exhausted their ingenuity with refined distinctions in this one physical sign, thereby only giving proof that there is more sense in their fingers than in their heads. I have formerly had occasion to explain the elements and the chief varieties of the pulse. Depending as it does on the size and condition of the arterial tube, and on the quantity and quality of the blood, as well as on the motions of the heart, it may be varied by many circumstances that do not affect that organ, and the motions of that organ may be variously intercepted or modified before they reach the artery, which is commonly felt by our fingers. But although these circumstances impair the accuracy of the positive indications of the pulse with regard to the heart, if we only take the trouble to analyse them, they may give us useful information, positive or negative, with regard to the condition of the circulation.

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* For the diagnostic properties of the different murmurs, see tables iii. and iv. the end.

The radial pulse, in general, represents truly the number of the heart's contractions; it never can exceed them; but when the heart acts very feebly, its pulsations may not reach the wrist; and when they are irregular in force, some may be propagated to it and others not, in which case the pulse intermits. On listening to the heart, the ineffective pulsations are heard, and they are often loud enough, but their character, together with the fact that they do not reach the wrist, may inform us as to the morbid condition of the organ on which they depend; and we may learn from the same examination, that however violent the heart may be seeming to act, it is defective in its power to propel the blood through the system. This inconsistency between its apparent and its effective power is often manifest on comparing the pulsations of the heart itself during a fit of violent palpitation with the comparatively weak pulse that reaches the wrist. These discrepancies do not, however, occur in every case; for in some individuals the radial pulse pretty precisely represents the number, force, and even the time of the contractions of the ventricles, however varying they may be. This it does in those individuals in whom the coats of the arteries are rigid and unyielding, and transmit unmodified each pulse-wave from the heart. Where, on the other hand, the arteries are thin, elastic, and imperfectly distended, they may soften the hardness of the heart's pulse, in its course through them, reduce its strength, retard its period, and if it be small, annul it altogether. Thus, with our ear at the heart, and our finger at the same time on the radial pulse, we may take a far more accurate survey of the condition of the circulation than we can by examining these parts separately; and the utility of this method is by no means confined to affections of the heart. Thus, in inflammatory fevers, the pulses at the wrist owe their hardness and strength in some measure to arterial tone or tension, for they are more instantaneously propagated from the heart, and *accompany* rather than follow the ventricular systole. In debilitated atonic states of the system, on the other hand, the radial pulse *follows* the first sound of the heart by a distinct interval, which is occupied by the transmission of the wave along the course of the comparatively lax and yielding artery; and it is in passing through such a tube that the pulses lose also their strength and hardness, and those that are weak may be altogether suppressed. Thus, independently of disease of the heart, a pulse that reaches the wrist tardily and weakly, is a sign of an atonic or asthenic state of the vascular system; whilst one that is transferred with celerity and force, equally indicates that fulness and tension of the vessels which is the chief character of sthenic or inflammatory conditions. I recommend this comparative examination as well worthy of attention, for I have often found it, as well as the character of the sounds of the heart, give positive practical indications in various diseases, when the pulse alone and other symptoms wore a doubtful aspect.

The character of the pulse may be very much modified by diseases permanently affecting the vital properties or mechanism of the heart and arteries. These varieties will be noticed as we treat of these diseases. I shall here only exemplify some of the different elements which are concerned in their production. A large strong heart acting with a prolonged first sound will cause a strong full pulse, which will be hard in proportion to the tension of the arterial coats. A large weak heart with a short first sound will produce a pulse which may be short or abrupt, but not strong, and it generally occupies an appreciable interval of time in passing to the extreme arteries. It may be small or large, according to the size of the artery and the general fulness of the vessels. Defects in the valvular apparatus of the left ventricle tend also to modify the pulse by altering the manner in which the blood is propelled from it. When the mitral valve does not close perfectly during the systole, part of the strength of this action must be lost backwards into the auricle, and the pulse will be weakened in proportion.—This regurgitation commonly produces a murmur, and where it is considerable, the pulse is also generally irregular. When the aortic valves are imperfect, the tension of the arteries is not maintained beyond the period of the ventricular systole; for when this ceases, the blood returns into the ventricle with a freedom proportioned to the insufficiency of the valves. This occasions a very peculiar character in the pulse. Owing to an increased thickness of the walls of the ventricle which commonly accompanies this lesion, the pulse is strong at the moment of the systole, but recedes instantly after, from the absence of the arterial tension, so that it has a jarring or jerking character which is very remarkable, and which often makes its motion visible, the artery starting into a slightly tortuous line at each pulse. This visible pulsation of the arteries has been described by Dr. Corrigan as a sign of incapacity of the aortic valves. But it is also seen in other cases in which the action of the heart is abrupt, and the arterial tension incomplete. Constriction of the aortic orifice diminishes the strength of the pulse, but, unless extreme, not its hardness and sharpness. Constriction of the auricular orifice is generally accompanied with great irregularity of the pulse.

Venous Murmurs.

I have already mentioned that sound may be produced in the veins, as well as in the arteries, by certain modifications in the current of blood through them. This fact was first announced by Dr. Ogier Ward, in March 1837. In the summer of the same year, I made a series of researches, which were afterwards repeated by Dr. Todd and myself, and I reported the results at the meeting of the British Association in the following September. The report was published in the Medical Gazette for Dec. 2, 1837, and will be found in the appendix. In 1839, Dr. Hope published observations on venous murmurs, corresponding in most particulars with our previous report.

Laennec, as well as all his successors, described loud bellows and musical murmurs as heard in the neck, and also in the region of the heart, which differ from the ordinary ones in being continuous, or at least remittent, instead of in pulses. The most common kind is a strong blowing or rushing sound, which, although continuous, is augmented at each pulse of the artery. This, from its resemblance to the noise of a toy, called in France a "diable," Bouillaud has termed "bruit de diable." In other cases it is whistling like the sighing of wind through a key-hole ; or like the humming of a gnat or fly, ("bruit de mouche,") or the singing of a tea-kettle. Sometimes it changes suddenly from one of these to another. After Dr. Ward's paper appeared, I was slow to believe that the venous current was strong enough to produce such loud sounds; but my doubts were entirely dispelled, when in my experiments I found that a very gentle stream of water falling only *guttatim* from the end of a thin caoutchouc tube, caused loud and exactly similar sounds when the tube was compressed by the end of the stethoscope. On trying the current in the jugular veins of the living body, I recognised the same sounds, and verified the observations of Dr. Ward, that they were arrested by pressure above the stethoscope—impaired or modified by efforts at expiration, by lying or stooping the head—and increased by inspiration, and by raising the head. The situation in which the venous murmur is usually best heard, is at the posterior edge of the right sternomastoid muscle, a little above the clavicle, which corresponds with the position of the external jugular vein. In anæmic subjects it may, however, be heard in almost every part of the front side of the neck, and is certainly often produced, independently of the pressure of the stethoscope. In several instances I have found it accompanied by a continuous thrill, sensible to the finger immediately above the sternal end of the right clavicle. This was first shown to me by Dr. Taylor of University College Hospital. Venous murmurs are not, as supposed by MM. Andral and Bouillaud, confined to anæmic or chlorotic subjects, for I have succeeded in producing them in many robust subjects, by various degrees of pressure by the end of a flexible ear-tube without its cone. Still it is when the blood is thin and the vessels ill filled, and the current consequently rapid under the influence of gravitation, of the suction of inspiration, and of similar causes, that the phenomena are best developed. Besides, in the neck I have often heard an obscure continuous murmur on either side of the upper sternum, in the region of the heart, and between the scapulæ: the latter situations have presented it in cases where there were other symptoms of impeded transmission of blood through the lungs or heart. Near the top of the sternum I have generally found a murmur in connexion with substernal aneurisms and other tumours which cause partial obstruction of either of the venæ innominatæ. I have met with a permanent venous murmur in several cases of contracted chest, after pleuro-pneumonia. Venous murmurs are less common in other parts besides the neck and chest, there not being elsewhere the rapidity of current necessary to produce them; but Dr. Hope states that he has heard them in the abdomen, and I have succeeded in developing them in the large veins of the extremities by changes of posture.

The variety of murmur called by Laennec musical, is now readily explained; it is chiefly heard in the neck, and has its seat in one or both of the jugular veins. The variations in tone depend not only on the pressure of the stethoscope, but on that of the muscles also, on the pulse of the adjoining arteries, and especially on the variations in the venous current from the respiratory movements. When once the murmur is pitched in a musical tone, the pulses and the breathing modify it into a regular series of notes or tones, such as those which Laennec and others have actually written in musical characters. Shifting the stethoscope or the position of the neck converts the notes into a continuous roar.

CHAPTER II.

FUNCTIONAL DISORDERS OF THE HEART.

Increased or inordinate Action and Palpitation.

Having said as much as our space would allow, on the physical properties and phenomena of the heart, and the modes of examining them, we now come to its special pathology—to trace the modes in which its functions and structures may be affected by particular kinds of disease, and the phenomena that it may present under these affections. The function of the heart is so complete in itself, and its mode of action so determinate, that we may well introduce its diseases by a notice of the disorders of its function, which may be independent of inflammation or change of structure, although these are not unfrequently their causes.

Functional disorders of the heart may be divided into those of *increased* action, those of *irregular*, and those of *defective* action.

The general symptoms of inordinate action of the heart are commonly either *palpitation*, which is the action of the heart perceptible to the patient, and is sometimes very distressing; or a feeling of fluttering, agitation, or anxiety, within the chest, without any distinct throbbing. There is frequently greatly increased action, without the patient being conscious of any palpitation: the feeling complained of is rather breathlessness, although the affection of the breath is only secondary. It is great violence, rather than frequency of the pulsations, that renders them perceptible to the patient: and if we take this character as constituting palpitation, we may, contrary to the statement of Laennec, have palpitation with a slow pulse. In fact, the *feeling* of palpitation may be induced by augmented sensibility of the heart or of the adjoining parts, where there is very little increase of action; and many sensitive and nervous persons can always feel the motions of the heart independently of any inordinate action. The feeling of palpitation cannot, therefore, be taken as a general measure of the action of the heart, but rather of its sensibility. When the sensibility is great, there may be pain with the palpitation; and this is sometimes very acute, being of the lancinating, constricting, or burning kind, shooting to the back, left shoulder, and arm, which is called *angina.* Sometimes it is duller, and more like the feeling of soreness. The pulses of an over-active heart are sometimes felt in some of the chief arteries more than in the organ itself: this is especially the case in the carotid and vertebral arteries, in the arch and descent of the aorta; hence the throbbing in the head, neck, at the top of the sternum, and in the epigastrium, which is sometimes as distressing as palpitation. We often observe that the chief force of inordinate action of the heart seems to be expended in the first parts of the arterial tube, and does not reach the extreme branches, the pulses in which are not unfrequently weaker than usual. For the same reason, also, whilst the head is hot and the face flushed, the extremities are often cold; or, if they retain heat, they are dry and unperspiring. Hence, too, if the inordinate action be prolonged, may arise disorders of the secreting and nutrient functions: the irregular distribution of blood overloading the vessels of some parts, and imperfectly supplying those of others.

On physically examining an excited heart, we find the impulse and sounds much increased, as after violent exertion. We feel it abruptly and forcibly knocking against the ribs; but if it be not enlarged, we can perceive that its stroke is short and circumscribed, like that of a moderate-sized body, and we find on percussion that the extent of dulness is as limited as usual. The first sound is shorter than usual, and so loud that it can often be heard without the actual contact of the ear or the stethoscope with the chest. The second sound is sometimes also loud; but, if the pulsations be very rapid, which they sometimes are by double or treble their usual rate, the second sound becomes indistinct, and a mere appendix to the first; the quantity of blood thrown into the arteries at each pulse being insufficient to make their valves flap freely and with force. When the action of the heart is increased in force more than in frequency, which occasionally happens in excitement from plethora and sthenic conditions of the vascular system, the impulse is strong, but less abrupt; the first sound longer, and the second sound louder, than where the pulsations are frequent. These characters are also permanently found in the pulsations of some enlarged hearts, but then there is more dulness than usual on percussion in the region of the organ. In violent palpitation, the enlarged pulsations of the arch of the aorta may be felt and heard loudly at the top of the sternum, those of the subclavians below the clavicles, and of the carotids in the neck, where also the jugular veins are often seen pulsating, from the retropulsive action of the right ventricle.

When the inordinate action of the heart is excited by transient causes, its duration may be temporary, constituting what are called fits of palpitation; and the heart, when permanently diseased, is frequently worked into these fits by slight additional irritations. They are often most painful and distressing; being attended with feelings of oppression, of extreme faintness, of indescribable agitation, &c.; sometimes of severe anginal pain, cutting or tightening across the chest, and sometimes shooting to the back, neck, and arms. They are in some cases aggravated by fits of spasmodic asthma, of which they may be either the cause or the effect. These formidable symptoms belong chiefly, but not exclusively, to organic diseases of the heart.

When inordinate action of the heart depends on inflammation increasing the irritability of its tissues, it is more permanent in its duration, partaking of the course of the inflammation. But as the inflammation does not affect all parts of the heart alike, some may become more irritable than others, and contracting more abruptly, or with greater force, may modify the character of the systole and its sound. This happens especially when the irregular irritability includes the muscular pillars which regulate the motions of the mitral valve. If these act too suddenly, but perfectly, the flapping commencement of the first sound may only be exaggerated; but if they act irregularly, so as not, with the pressure of the blood, to close the orifice of the auricle, the blood will regurgitate through it, causing a blowing murmur, which is generally heard best where the apex is felt beating. This regurgitation, if considerable, may develope another cause of excitement in the obstruction which it causes to the onward progress of the blood: this subject we shall notice under the head of valvular disease.

Irregular Action of the Heart.

The irregularity may be either in the *rhythm* or *period* of the pulsations, or in their *strength*, or in *both*.

Slight irregularity in the *rhythm* of the heart is by no means uncommon in persons who are weak or nervous. Thus we often find a pulse now and then occur sooner or later than in the natural order, constituting *reduplications* or *intermissions* of the pulse. Such phenomena are very common in those who are suffering from much bodily fatigue. They may occur quite independently of disease of the heart, although they are also frequent concomitants of it. An intermittent pulse may be said to be constitutional in some persons; but they generally have other signs of a weak or an easily disordered circulation. When there is no disease of the heart, the sounds of the irregular pulsations are natural, except that those which occur before their time may be shorter and more abrupt, and the retarded ones are stronger and more prolonged than usual, which are the simple results of the different quantities of blood which the contractions have to propel. In the absence of signs of other disease which may cause it, simple irregularity of the pulse may be considered a result of defective tone of the heart and vessels, and may sometimes be taken as an indication for the use of animal food and tonics.

Irregularity in the *strength* of the heart's pulsations commonly depends on organic disease. It may be more evident in the arterial pulse than it is in the region of the heart, for, being there reduced by distance, the weaker beats may be scarcely or not all perceptible; and the artery, consequently, often has a pulse much less frequent and regular than the heart. Many of the instances that have been recorded of extremely slow pulse are probably of this kind. I have met with several cases in which the pulse at the wrist was below thirty, and irregular, when that of the heart was sixty or ninety, and pretty regular as to rhythm, but varying much in strength, so that the strong beats only reached the wrist. But it is more common for the rhythm to be also disordered, so that on listening to the heart, instead of beats at regular intervals, they occur in all varieties of measures, very unequal both in time and strength: and at the wrist the inequality is perhaps still more evident. Amidst all this irregularity, when perhaps no three consecutive pulses are of the same period or strength, there is very often something like an attempt at order, every third, fourth, or fifth beat being stronger, and followed by a longer interval, so that a sort of tattoo measure is more or less kept up. In persons whose pulse only occasionally presents these irregularities, we may sometimes observe another curious phenomenon: the heart, which was beating regularly at sixty, will suddenly double or treble its number of pulsations, still preserving its regularity at 120 or 180, producing the feelings of flurry, oppression, faintness, or angina, that have already been noticed as attendant on inordinate action. After a while the pulsations may, as suddenly as they quickened, fall back to their slow regular standard. But sometimes, instead of this sudden and complete change of measure, the two rates of pulsation seem to be variously mixed; three or four beats of the quick movement being followed by a strong one of the slow, and a pause, or some other such combination. This is a common kind of the permanently irregular pulse that we meet with in certain forms of organic disease of the heart, particularly those affecting the left auricular valve; and in the tendency to periodicity in the recurrence of the stronger and slower beats, we still see the remains of a natural rhythm, which prevails most in the more tranquil conditions of the circulation.

On listening to the pulsations of a heart acting irregularly, we find a good illustration of the principle, that the character of the sounds depends in great measure on the mode of action. Here is the same heart giving in succession a considerable variety of sounds, the quick pulses being short, loud, with little impulse, and without a second sound; and the slower ones every now and then coming in with a longer heavier sound, strong impulse, and with a second sound; whilst the valvular disease that is frequently also present in these cases may add a murmur, which modifies some or other of these sounds, according as it may occasion the blood to pass in an unnatural manner. Commonly the short quick pulses, belonging to the high rhythm, prevail the most, the strong pulsations being only occasional; but sometimes, in a pulse which is slow and pretty regular, there will be now and then two or more minor quick pulsations, which often do not reach the pulse at the wrist. These being short, and unaccompanied by a second sound, were supposed by Laennec to be repetitions of the second sound, which he referred to the contraction of the auricles. But they may often be felt in the carotids, and are thus proved to be minor ventricular pulses; in fact, they exactly resemble, and are examples of, the short rapid pulsations of a high rhythm. When this rhythm prevails altogether, to the amount of 160 or 200 in a minute, there is such a vibratory commotion in the region of the heart, that one is almost tempted to suppose that the two ventricles are acting alternately; but this supposition is hardly tenable, when we consider that their fibres are in great measure continuous, and not adapted for separate action. In judging of the size or strength of a heart acting irregularly, we must attend chiefly to the strong slow pulsations, which best represent the true capacity and properties of the organ; for the frequent rhythm, with short sound and no impulse, may take place in any kind of heart, large or small, being perhaps produced by a twitching or quivering movement of the fibres, rather than a real and complete systolic action. The strong pulsations also may inform us, by the murmur which may accompany them, as to the condition of the orifices. Lesions of some part of the valvular apparatus of the heart are generally the foundation of permanently irregular action, although its immediate occurrence may be determined by more transitory causes. Of many cases of habitual irregularity in old people, I have never failed to find signs of disease of the orifices or valves to a greater or less extent, although in several instances the constitution had, in a measure, adapted itself to the anomaly, and the individuals suffered only occasionally from shortness of breath, palpitation or fluttering at the heart, faintness, or some similar symptom of inefficient central circulation. But these persons cannot undertake great exertion without suffering much from it; and attacks of catarrh, or such affections, which might be trivial in others, fall heavily on them. I have met with several examples of irregular action of the heart beginning after unusual bodily exertion and mental excitement, chiefly in stout persons, and in those of a gouty habit. In most of these the irregularity subsided in time, after quiet moderate living, and a due action in the secretions; or more rapidly on the appearance of gout in an extremity.

Defective Action of the Heart.

The action of the heart may fail in force, or in frequency, or in both; and its failure, in either respect, when extreme, produces the phenomena of *syncope* or *fainting*.

The general symptoms of syncope are, feelings of sickness, giddiness, swimming in the head, noise in the ears, indistinct vision, sudden pallidity or change of the countenance, occasionally with quivering of the lip, chattering of the teeth, involuntary groaning, slight convulsion, and ultimately loss of consciousness and apparent death, which may last for some seconds, minutes, or even hours. Restoration is often accompanied by the breaking out of perspiration, eructation of wind, vomiting, slight convulsions, or palpitation; and sensations far more unpleasant than those which preceded the loss of consciousness, and which some have described as rather agreeable than otherwise. The radial pulse is either very weak or altogether imperceptible during syncope. The sounds of the heart are also commonly feeble, very short, and generally without a second sound; sometimes they are frequent, in other cases very slow, and they are generally irregular or unsteady. I have never met with a case of syncope in which they were inaudible; but we can readily conceive that pulsations may take place with so small a force as to produce no audible sound, and yet to propel the blood enough to preserve life. In some of my experiments on animals, slight contractions recurred for some time after all audible sound had ceased; and in one instance the auricles alone continued to act alternately for several minutes after the ventricles had lost all motion.

But the action of the heart may be defective, yet short of the degree which causes the phenomena of syncope. This defective action may be caused by the same circumstances which occasion syncope, in which case it may be of temporary duration; but it may also arise from a more permanent weakness or defective contractile power of the heart. Under any of these circumstances there will arise more or less of the general effects of imperfect or languid circulation of the blood. Such are coldness of the extremities, often attended with flushing or a feeling of heat in the head and face; a general feeling of languor, with pains in the back and limbs; shortness of breath, and fainting on slight exertion; œdema of the feet, sick headaches, a loaded tongue, weak digestion, constipated bowels, and a defective or disordered condition of all the secretions. The pulse is weak and small, and so is the impulse in the region of the heart, although it may be abrupt; and the accompanying sound is pretty loud, but short. The action of the heart may be either frequent or slow; but it is most readily excited, by exertion or any other cause, to palpitation or other kinds of irregular inordinate action. It is often this palpitation that alone draws attention to the heart; and not unfrequently a lowering treatment has been adopted to reduce this inordinate action, when it has all the while been the result of the mere weakness of the organ, which struggles at a task to which it is unequal.

The general symptoms of many organic diseases of the heart are also those arising from deficient effective action of the organ, although its apparent power may at the same time be very great. It is of great importance to hold this view in relation to the treatment; for in many cases of such affections, as well as in others in which inordinate action arises from want of power rather than from its excess, more benefit may be derived from a judiciously directed tonic plan, with especial precautions not to overtax the weak organ, than from measures directed against its occasional excitement.

Angina Pectoris.

Exalted sensibility of the nerves of the heart causes the phenomena described under the names, *angina pectoris*, *syncope anginosa*, *sternalgia*, &c. Pain in the region of the heart, accompanied by a feeling of faintness, is in truth rather a symptom than a disease; and it may occur in various degrees in various affections of the heart and great vessels, functional as well as organic. The kind of pain, cutting, piercing, tearing, burning, crushing, shooting to the back, arm, and throat, and so forth, as it is described in different cases; the suddenness of its attacks, and the temperament of the individuals in whom it occurs independently of organic disease, evidently refer it to the class of neuralgic pains. I have known it in a very severe form attack patients who had before been sufferers from tic douloureux. It is commonly associated with organic lesions, especially, but not exclusively, that affecting the aorta and its valves; but such affections frequently exist without it. So, on the other hand, anginal or neuralgic pains sometimes occur in persons who have no organic disease; thus resembling other forms of nervous disorder, which, although occasionally excited by the irritation of bony deposits, or other permanent lesions, may arise from an excessive sensibility developed by more transient causes. The circumstances which generally determine the immediate attack of a paroxysm of angina are those which excite the heart's action, such as walking quickly up a stair, up a hill, or against a strong wind, strong mental emotions, distension of the stomach, and the like.

CHAPTER III.

INFLAMMATORY DISEASES.

Pericarditis.

Inflammation of the heart produces different symptoms and consequences, according to the structure which it chiefly affects. It commonly attacks the serous and fibrous membranes, either covering the exterior of the organ, or lining its interior, and by their folds forming the valves; more rarely it affects the muscular fibres, or the cellular texture which unites them. Hence the division of inflammation of the heart into the species, *pericarditis*, *endocarditis*, and *carditis*. The two first are commonly combined, but not constantly; and we may well consider them separately.

Inflammation of the pericardium, as of the pleura, tends to produce an effusion of serum and coagulable lymph on the free surfaces of the membrane; by these products, as well as by other changes of the vital and physical properties of the heart, pericarditis developes various symptoms and signs by which we may ascertain its existence.

I do not consider it necessary to describe at length the various morbid appearances which are found after death in the inflamed pericardium. They vary like those of pleurisy, and I refer to the descriptions which I have given of them. There is this difference, that the motion of the heart being constant, and not like that of the lung interrupted by the effusion, it in various ways modifies the deposition of lymph, according to the quantity of accompanying liquid; and thus are produced those transverse ridges and furrows, honeycomb or reticulated, and shaggy or villous appearances, which are seen in the lymph of an inflamed pericardium, and which Laennec imagined to be a peculiar kind of new-structure. The liquid may be serous, sero-purulent, or purulent; with all the varieties that are found in the liquid of pleurisy.

The general symptoms are often very obscure. There may be pain below the left breast, shooting to the sternum, back, and left arm; perhaps with tenderness on pressure on or between the cartilages of the fourth, fifth, and sixth ribs, or on the epigastrium; but all such pain or tenderness is sometimes entirely absent, and is more commonly produced by pleurisy than by pericarditis. Neither is dyspnœa nor palpitation always present, although they are sometimes extremely distressing, and give the patient's countenance an expression of anxiety, uneasiness, or restlessness, which is very characteristic, and may be perceived even when the patient does not acknowledge the existence of the sensations. I have often known pericarditis to exist without producing in the chest any unusual sensation whatever; and even the pulse in the first stage has been below its usual frequency. Besides this, these symptoms are occasionally disguised by delirium, or a state of incoherent stupor, which draws all the attention to the brain, although it is only secondarily affected. Under these circumstances it is not wonderful that the existence of the disease should have been often overlooked, and known only through an examination after death, or by the production of a permanent organic disease, which is more unequivocal in its symptoms.

But pericarditis cannot exist for any length of time, and certainly not to a dangerous extent, without producing physical signs that are quite characteristic. That these have often escaped notice, even of auscultators, is because they have not been sought for. It is of great importance, therefore, to examine the region of the heart in all cases in which there is the probability of its being affected, whether any symptoms have manifested themselves or not. The cases in which we should especially be on the look-out for the signs of pericarditis are those of rheumatism in all its forms, but particularly the acute kind, which is generally called rheumatic fever; the subjects of mechanical injuries, or of pleurisy or pneumonia affecting the left side; and all doubtful instances of difficult breathing, especially when accompanied by œdema, general or partial. Of the cause of pericarditis, rheumatism is by far the most common; but it still more frequently produces endocarditis. In fact, although I do not agree with Bouillaud's opinion that these inflammations are essentially a part of rheumatism, I can confidently state that I have found signs of endocarditis or pericarditis, or both, to a greater or less extent, in fully three-fourths of the severe cases of rheumatism which I have examined in the last six years, although in little more than half that proportion was there any complaint of pain in the chest, palpitation, or dyspnœa. That the membranes of the heart are frequently affected in cases of rheumatism, has been long acknowledged by English practitioners.

The first physical sign of inflammation of the membranes of the heart is an increased loudness of the natural sounds, with an impulse stronger and more abrupt than usual. The pulsations are generally but not always accelerated; sometimes they are slower than usual, and then they are stronger; and the first sound, although loud, is prolonged even when there is no murmur. Sometimes there is now and then a reduplicated pulse, or some other slight irregularity; but the beats are for the most part regular, and they owe their loudness to their increased vigour. In this state of the signs the inflammation may be in the pericardium, endocardium, or both, without exhibiting anything distinctive of its seat for from twenty-four to forty-eight hours, and may sometimes be removed by general and local blood-letting, without proceeding further. But in that time, and perhaps in less, new signs commence, which announce changes distinguishing the seat of the inflammation. Those of endocarditis are various murmurs, which are commonly the first to show themselves; but we must consider them afterwards. Those of pericarditis are various sounds of superficial friction, which are quite characteristic. At first this sound is soft and rustling, like the rubbing together of two pieces of paper or silk stuff; and it may accompany only part of the natural sounds, from which, however, it is obviously distinct, in being much more superficial. It is generally heard first about the middle of the sternum, or to the left of it, corresponding with the base of the heart, or the attachment of the auricles. It afterwards increases in loudness and duration, being heard beyond the immediate region of the heart, and accompanying not only the periods of the natural sounds, which it disguises, but also the interval between them. It thus gets a sort of continuous jogging rhythm, corresponding with the movements of the heart, which is like that of the saddle when one rides on horseback; and when, as it generally happens, the friction-sound becomes harder, and more like the creaking of leather, its resemblance to the noise of a new saddle is quite ridiculous. In some cases the noise is crackling, like that of crumpling dried membrane or parchment. We owe the discovery of this valuable diagnostic sign to M. Collin; and it has more recently been described by Reynaud, Drs. Stokes and Watson, and others. I have had many opportunities of observing it in its different modifications, and of verifying its accuracy by examination after death, during the last ten years. Laennec and Bouillaud did not recognise it, even after M. Collin had described it in 1824, and it was unnoticed by Dr. Hope in his edition of 1835. They must have mistaken it for the double grating murmur of diseased aortic valves, from which, however, its more superficial, diffused, and less regular character, easily distinguish it. These friction-sounds are certainly caused by the rubbing of lymph on the pericardium proper, and on its sac.* The natural sounds of the heart are completely disguised by a loud friction-sound; but they may sometimes be heard at the top of the sternum, and in the carotid arteries, and they will be often attended with a murmur; this depends on the simultaneous existence of endocarditis, and will be noticed presently.

The pericarditic friction sound does not commonly continue for many days in succession. Either the lymph is absorbed, or it is converted into false membrane, which forms a bond of adhesion more or less perfect between the heart and the sac, in which case the sound gradually ceases; or if the inflammation continue in a low degree, serum is poured out in such abundance, that the heart moves free in the distended sac, and no longer rubs against it. In either case the friction-sound becomes less constant, accompanying only the stronger pulses, and gradually ceases. In cases of adhesion, it is commonly heard longest below the left breast; and when it is terminated by liquid effusion, it generally continues latest near the sternum, corresponding with the base of the heart, which is less easily separated from the sac than the apex. In the latter case, change of posture, as by leaning forward, will also sometimes reproduce it when it has ceased. I have known it in a few cases to continue for more than a fortnight in conjunction with the signs of liquid effusion ; for this effusion does not always proceed to an extent sufficient to separate the heart from its sac, particularly at its anterior surface.

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* Some experiments performed for the British Association by Drs. Clendinning, Todd, and myself, with a view to determine the physical cause of the friction-sound, have led us to the conclusion that inflammatory injection of the pericardium is insufficient to produce it; but the smallest effusion of lymph, and even slight ecchymoses, or effusions of blood under the pericardium proper from acupunctures, may cause distinct rustling or rubbing sounds. A very good example of the leather-sound was presented by a heart which was found to be covered by a thin film of soft lymph.

The effusion of serum in the pericardium produces dulness on percussion in the region of the heart; whilst, if copious, by removing the heart from the walls of the chest, it generally renders the sounds more distant, and more or less impairs the impulse. The dulness may be limited to an area of two or three inches at the lower part and to the left of the sternum; or, in case of extensive effusion, it may occupy the whole front of the left side of the chest, even as high as the second rib, and extend even to the right of the sternum. There being no sound of respiration, and sometimes a prominence of the ribs in these parts, the case might be taken for pleuritic effusion, but that the sounds of respiration and percussion are still good in the back and below the axilla, and are not materially varied by the change of posture. There does not seem to be any fixed rule which regulates the displacement of the heart by liquid in the pericardium ; for although this organ is commonly displaced in proportion to the quantity of the effusion, so that the friction-sound is stopped, and the impulse and natural sounds are rendered distant, yet I have met with cases in which extensive dulness on percussion indicated copious effusion, and the friction sound and impulse still continued strong at the sternum. We may discover when the sounds are rendered weak merely by displacement, on listening to them at the top of the sternum, and in the carotid or subclavian arteries, where they may be heard of their usual intensity. This enables us to distinguish this case from that of a greatly enlarged heart acting very feebly : in this instance there would be extensive dulness on percussion, with weak sounds and impulse, but the sounds would also be weak in the course of the arteries. When the liquid in the pericardium is scanty, it may only partially intercept the impulse, and modify rather than obscure the sounds. Thus in the standing or sitting posture the impulse at the apex is diminished, and the sounds assume that more drum-like character that I have before described as the effect of an intervening layer of liquid ; whilst at the sternum both sounds and impulse may be as usual. In the supine posture similar differences may be presented in an altered relation; the drum-like sounds and diminished impulse corresponding with the dulness on percussion.

With all the signs of extensive disease that I have described, the general symptoms may be combined in most varying and uncertain proportions; being sometimes well marked, and necessarily drawing the attention of the practitioner to the seat of the disease; in other cases being so trivial or equivocal as scarcely to be acknowledged, even by the patient. Some feeling of dyspnœa or faintness, especially on moving, is the most constant symptom; and this is generally accompanied by irregularity of the pulse. The contractility of the heart, which was in the first instance exalted by the inflammation of its membranes, ultimately becomes impaired; the action loses its rhythm, becomes sometimes palpitating, sometimes defective, and verges on syncope; syncope, in fact, sometimes does occur, and may suddenly terminate the patient's life. But this is by no means a common case. I have met with but few cases of pericarditis which first attacks a sound heart, proving fatal in its acute or chronic form; but I have known several partially or apparently recover from a first attack, after some months or even years exhibit signs of organic disease of the heart, and ultimately sink under a subsequent attack of pericarditis supervening on this. A large proportion of cases, even where the disease was very extensive, recovered entirely, as far as their subsequent history was ascertained. I do not, therefore, look on pericarditis as so formidable a disease as it has been generally considered to be. In fact, its existence has hitherto been known chiefly by those instances in which it has either proved immediately fatal, or has become conjoined with serious organic disease, which constitute but a small part of the cases which we can detect by physical diagnosis.

The course of pericarditis is, like that of pleurisy, very much determined by the nature of the effusion. When this is moderate in quantity, and composed equally of serum and healthy lymph, it may all be absorbed, and the cure completed in a short time. Such we may conclude to be the case when the friction-sound and dulness on percussion last only a few days, and no sign of disease of the heart remains. When the lymph is more abundant, adhesions will form between the heart and pericardium; and the character and effects of these adhesions will vary mainly according to the quantity of the liquid effusion, and the manner in which this protracts the period of their formation. If the liquid be moderate in quantity, or soon diminish so as to bring the lymph early into adhering contact before its plastic qualities are impaired, and whilst the heart is still vigorous and free in its action, the adhesions formed will then be loose and mobile, composed of yielding cellular membrane, which may very little, or not at all, interfere with the motions of the organ. Such adhesions have often been found after death in cases where their existence had not been suspected during life, not having in any notable degree impeded the heart's action. But if the liquid effusion be abundant, and persist long, there is great fear that the lymph effused will not be brought into adhering contact until its vitality is impaired, when it is capable of forming only denser and more fibrous adhesions, disposed to contract further, and when, the action of the heart being feeble and irregular, it cannot adapt them to its healthier and freer motions; in such cases the adhesions formed, whether partial or universal, will for ever shackle the motions of the heart, and constitute a serious form of organic disease. Such adhesions are most injurious when they confine the apex, naturally the most moving part: they do less injury when they attach the base only; but in this case they are often combined with internal organic disease, arising from endocarditis. Not only the quantity of the liquid effusion, and the consequent slow formation of the adhesions, may conduce to their injurious character and effects, but probably in some measure the nature of the inflammation also.

From what I have just said, it may be inferred that *adhesions of the pericardium*, according to their character, may or may not produce signs by which they may be distinguished. When they are mobile, and composed of yielding cellular texture, they do not alter the positions, motions, or sounds of the heart, and therefore yield no signs. When, in consequence of their closeness and rigidity, they do interfere with the heart's motions, and thereby irritate it into inordinate action, it may still be difficult to distinguish this inordinate action from that from other causes, so long as the heart is nearly covered by the lung. I have sought in vain for the "jogging or tumbling" motion which has been stated by Dr. Hope to be characteristic of an adherent heart. Such motion results from irregular action, often exists without any adhesions, and in some measure depends on the motions of the lung differently affecting successive irregular pulsations. But it often happens in case of close adhesions, that prior to their formation the pericardial sac has adhered in its distended state to the walls of the chest at the left of the sternum, so that when the heart also adheres to the sac, it constantly pulsates in close contact with these walls. This combination of circumstances gives us very appreciable signs. In the first place, the motions of the heart may be seen and felt much more plainly and widely than usual, drawing in the intercostal spaces at each systole. Then these motions, instead of being, as usual, intercepted by the expansion of the lung in a full inspiration, are always close to the walls of the chest; for these walls, instead of, as usual, rising from the heart upwards and outwards at each inspiration, carry the heart with them in all their movements. Under these circumstances, therefore, there will be, proportioned to the adhesion and the size of the heart, a space in which the pulsations are always felt, and the sound on percussion is always dull in every stage of respiration, and in every posture of the body. If we compare this with what I have before said of the shifting, varying force and position of the pulsations and the sound of percussion in the region of the heart in health, we can understand the difference; and the distinction extends also in part to other diseases which enlarge the heart, but do not fix it to the walls. When the heart is thus generally adherent to the pericardium, and this to the diaphragm and walls of the chest, the enlargement of the organ, which very commonly ensues, cannot readily take place as usual, downwards, and to the left; but it proceeds upwards and downwards, carrying with it the walls of the chest, to which it gives a remarkable projection about the ends and cartilages of the middle ribs. I have seen this accompanied by a retraction or hollow at the epigastrium. In other cases again, from the adhesions being more partial, the enlargement may take place laterally, as described by Dr. Hope, and bring the pulsating apex of the heart far to the left side. Various other changes of position may arise from different circumstances, especially when the pericarditis has been conjoined with pleurisy.

Endocarditis.

Inflammation of the internal membrane of the heart—*endocarditis*, as Bouillaud has very well named it, is a frequent but not a constant concomitant of pericarditis. Nor is it wonderful that inflammation should readily spread from one surface of the heart to the other, when we recollect how near they approach at the auriculo-ventricular and arterial orifices, where they are separated only by a fibrous structure of more strength than thickness. Now this fibrous structure is itself especially obnoxious to rheumatic inflammation, and it is chiefly by inflammation of this kind that both surfaces are simultaneously attacked. Pericarditis, from other causes, may exist without any inflammation of the lining membrane; and endocarditis, when it exists independently of rheumatism, may continue long without affecting the pericardium; the inflammation in these cases being chiefly confined to the serous membranes, and neither affecting nor traversing the fibrous tissue between them.

The endocardium is placed in very peculiar circumstances with regard to the effects of inflammation upon it. It is a serous membrane, and the product of its inflammation might be expected to be like those of the pleura, or any other serous membrane, lymph and serum. But as we found that the continual motions of the heart give a peculiar form and character to such products in the pericardium, so these motions, and those of the blood, must be continually acting on those of the endocardium; in fact, they must be continually swept away; the serum entirely, and the lymph also, except in circumstances very favourable for its concretion and adhesion to the secreting surface. It is no wonder, then, that endocarditis should not have been readily recognised; since, in many instances, the traces by which inflammation is known to have existed could not remain to indicate its seat. So we may be prepared to expect that the anatomical characters of endocarditis are far less obvious than those of most other inflammations. We do, however, not unfrequently find in subjects dead of rheumatic and other inflammations of the heart, or of older organic disease, indubitable traces of inflammation of the lining membrane. Thus, especially at the left auriculo-ventricular ring, and in the membrane lining the auricle near it, as well covering the laminæ of the mitral valve as in the aortic valves, especially at their attachments and *corpora arantii* in their margins, we meet with striated or punctuated patches of vascular redness, sometimes accompanied by an inequality, roughness, or softness of the membrane; occasionally, with soft thickening, or distinct films of soft lymph upon it. In some cases we find apparent abrasions or distinct ulcerations of the membrane; and to the edges of these, or to any roughness or irregularity which the valves or lining membrane may present, are attached little soft fibrinous bodies, generally of a ragged conical shape, and more or less tinged with blood, which have obtained the name of *vegetations*. These all are probably effects of the more acute form of inflammation. But the same parts often present changes which, in other structures, we are accustomed to refer to chronic inflammation, such as tough thickening, with partial puckering, induration, and even ossification, of the lining membrane, or of those folds of it which form the valves, and especially of the fibrous tissue that is a strengthening network in them. The tissues thus changed are liable to rupture, irregular distension, and other accidents which we shall have to notice under the head of Diseases of the Valves.

The general symptoms of endocarditis are still more obscure than those of inflammation of the pericardium. There may be fever with increased action of the heart, sometimes with irregularity, more or less of the feelings of palpitation or faintness, oppression or agitation, and pain, heat, or soreness, referred to the sternum or epigastrium; but most of these may be absent, and associated as they often are with general rheumatic fever and pains, and occasionally disguised by sensorial disorder, or gastric and hepatic derangement, the secondary effects of the deranged action of the heart, the symptoms which should fix attention on this organ in particular, are very frequently overlooked, or referred to the convenient and satisfactory category of *general irritation*.

If we examine the pathological effects of inflammation of the lining of the heart on the tissues of the organ, we see that it must do more than produce general irritation. If it be equally diffused over every part of the interior of the heart, it might, in the first stage, cause a general increase of irritability and action; but we have seen that it commonly prevails most in particular parts, especially at the aortic and mitral orifices; and we must suppose that it will modify the vital properties of these parts more than of others; there will be a spasmodic abruptness in the action of the muscular fibres there, or a permanent contraction of those fibres at the arterial orifices which are endowed with tonicity;* besides more or less thickening from the effusion of serum and soft lymph into the looser parts, as between the layers of the valves. These changes of properties vary with the progress of the disease. If the inflammation have continued for some length of time, the over-excitement of the contractile fibres leads to their exhaustion, weakness, relaxation, and ultimately to a change in their structure; and the softening and effusions may lead to stretching, ulceration, rupture, or in time to harder thickening, contraction, and adhesion together of the looser and more membranous parts.

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* Arteries gradually contract on the application of galvanism or of a mechanical or chemical irritant to them. It is most probable, therefore, that the fibres.

This brief sketch of the pathological effects of endocarditis, on which, had we time, we might enlarge with advantage, may suffice to render intelligible to you its physcal signs, which I think I may describe with some confidence, having narrowly examined them in a good many cases, and traced them in a few to the conditions disclosed after death. They are often combined with those of pericarditis, which may come on after or before them, and always very much mask them. Like in pericarditis, the pulsations are not always more frequent in the first instance, but the sounds are louder, and the impulse is stronger. In a few examples this has been all, and the loudness and strength have in the course of a few days subsided. But more generally the first sound becomes prolonged and double, or attended with a slight roughness, and eventually lengthened out into a blowing or grating murmur. This announces a modification in the current of blood in some part of the heart, and if we attend to the character of the sound and the situation where it may be best heard, we may with tolerable precision determine in which part of the interior of the heart it is produced. If it is heard best an inch or two below the left breast, where the apex beats, and is heard but little or not at all above the upper half of the sternum and in the carotid arteries, it is produced by regurgitation through the mitral valve, the closure of which the irregular spasmodic action of the fleshy columns renders imperfect. The nicely-adjusted mechanism of this valve requires, for its closure, that there be an equal drawing of all its cords, to spread the membranous laminæ against which the blood presses; and we can now see how either the excessive or the defective drawing of some by the spasmodic, or by the weakened contraction of their fleshy columns, would throw these laminæ into wrinkles or loops,

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in which this tonic contractility resides, would be more irritable and contract more under the first influence of inflammation.

forming little chinks, through which the blood may be squeezed in a sonorous jet at each contraction of the ventricle.

If, again, the murmur is distinct along the upper half of the sternum and in the carotid arteries, where it commonly has a rougher, more grating character, we may conclude that it is produced in the aortic orifice. It implies the presence of a partial obstruction to the current, formed by swelling or deposit of lymph on the valves, or by the tonic contraction of certain fibres, all results of the partial endocarditis. In these cases, the clear flap, which constitutes the second sound, is often also impaired, being dull or double; and is sometimes accompanied, or even replaced, by a short grating sound, which is heard best about the middle of the sternum, and may sometimes also be heard in the carotid arteries. These altered sounds arise from changes in the arterial valves, which, being thickened by deposit on them, or by infiltration between their laminæ, cannot flap clearly, and may not perfectly close, but leave a little chink between their edges, through which the blood regurgitates with a slight grating noise on the diastole of the ventricles.

There are few cases of acute rheumatism which do not, during their course, present some of these signs, which sometimes are transient, and cease with the fever, but more commonly continue for a long time after, and often become permanent. As far as my observation goes, I should state, that those indicating the affection of the aortic orifice are less enduring than those of the mitral orifice; but, whilst they last, they are commonly attended with more constitutional disturbance, and give to the pulse a sharp jerking character.

Endocarditis may cause many of the signs and pathological effects of valvular disease, and for this obvious reason, that this inflammation constitutes or induces valvular disease in its several varieties, and either in an acute or in a chronic form is its principal cause. So also endocarditis is often excited during the course of valvular disease or other organic lesions of the heart, causing considerable aggravation of their symptoms, generally increasing them, and hastening their fatal termination. We shall have to notice these effects under the head of diseases of the valves.

Carditis.

I shall say but little on the subject of *carditis* proper, or inflammation of the substance of the heart; for its occurrence is rare, distinct from the membranous affections. In cases of pericarditis and endocarditis with disease of the valves, I have not unfrequently found portions of the muscular tissue of a darker colour and more or less softened, which probably is the effect of inflammation; but I am by no means confident of this. The bright red colour of some hypertrophied hearts is considered by Dr. Elliotson to be inflammatory. A few instances are on record, by Corvisart, Latham, and others, of pus being found between the muscular fibres; and Bouillaud describes other conditions which he supposes to be the effect of inflammation, especially a gray and a yellow softening. It appears to me probable that inflammation may lead to these conditions in some instances; but Dr. Copland has seen them (and so have I) in various cachectic states of the body, where there had been no signs of inflammation, but where all the tissues were more or less discoloured, the blood being apparently in a diseased state. To judge that the tissue of the heart is especially diseased, we must see that it differs much in appearance from the other muscles of the same subject. We find, on comparing the same muscles in different subjects, a remarkable variety of colour; and in some there is no freshness in any of the muscles, but all are pale, and verging on a pinkish drab or dingy brick-colour. Where this is not the case, a pallid yellowish appearance of the substance of the heart is not at all an uncommon accompaniment of other lesions of the organ, especially adhesions of the pericardium and accumulations of fat; but I should be inclined to refer this to an altered state of the nutrition of the organ, owing perhaps to partial obstructions in the coronary vessels, rather than to the immediate influence of inflammation.

The symptoms and signs of carditis, in the cases on record, have been pain in the region of the heart, with fever, dyspnœa, extreme anxiety, agitation, and perhaps delirium; rigors, palpitation violent at first, passing afterwards into fluttering, fainting, and irregular movements; and in a very few days death ensues. In simple and general carditis, I presume the sounds would be altered in intensity rather than in character; being louder at first, and soon becoming weak: but it is probable that generally some parts would be more affected than others, or the apparatus of the valves would be also involved; in which case there might be produced murmurs of various kinds. It is scarcely possible that acute inflammation can continue in a considerable part of the substance of the heart without interrupting its function, and rapidly destroying life; but it is conceivable that a small portion might exist, and even form an abscess, or, in a more chronic form, lead to a change of structure. Whether the yellow fibrous transformation of the muscular substance that is occasionally met with be the result of a process of this kind, I have not had a sufficient number of facts to enable me to determine; but I have seen, in cases of long-continued disease of the heart, among other changes in which an inflammatory process could be recognised, a developement of whitish fibres, and even laminæ, in the fleshy substance, which had much the appearance of the commencement of fibrous degeneration.

I shall return to this subject under the head of Hypertrophy, which, with Dr. Elliotson, I believe to be often akin to inflammation.

CHAPTER IV.

STRUCTURAL DISEASES OF THE HEART.

When we speak of the symptoms of organic disease of the heart, we include the phenomena of its function, disordered in consequence of an injured mechanism or an altered tissue: these, as far as regards their physiological relations, may in great part be also produced by other causes less permanent than those of diseased structure; and it is only in case of their great extent or permanency that they can be judged to indicate organic disease.

Hypertrophy of the Heart.

The *muscular substance* of the heart may become variously altered in quantity, form, and quality; and its several changes may in different degrees be traced to causes affecting either the motory functions of the organ or its nutrition, or (as is more commonly the case) both together. The most common and familiar of these affections, hypertrophy and dilatation, opposite as they seem, often arise from similar causes, but operating on a different condition and quality of tissue. When from any cause, whether the quantity or quality of the blood or the irritability of the fibres, the heart is excited to excessive action for a considerable length of time, and the nutrient function of its vessels is not impaired by a general cachectic or chlorotic state, like any other highly-exercised muscle, it increases in substance, it becomes over-nourished or *hypertrophied:* and there is, in the case of the heart, an additional reason why increased action should eventually lead to hypertrophy—namely, the direct relation of its nutrition to its own action. When speaking of inordinate action, I had occasion to observe that the great force of the pulsations is commonly expended on the first parts of the arterial tube; so that whilst the throbbing is strong in the aorta and immediate branches, the pulse in the radial and other distant arteries is often uncommonly weak. Now the coronary arteries, which supply the heart, are the first to profit by this partial force; and whilst they are furnishing the tissues of the heart with the nutrient fluid, in force and abundance greater than usual, distant parts may be languishing for lack of a due supply. Thus increased action may extend from function to structure, and continued excitement becomes perpetuated by augmented substance and strength. But if the blood be defective in its nutrient quality in respect to muscular fibre, as it is in various cachectic conditions of the body; or if the strength of the heart itself be unequal to propel the mass of blood on which it endeavours to contract, excited action will not be backed by increased nourishment; the contractions, although still quick and abrupt, will have no extent of force, and the fibres, unable to expel the mass of blood to the usual amount, will become permanently extended—spread abroad: the walls will be thus *dilated*, and the cavities enlarged. There are other causes of dilatation, which we shall consider presently. Again, these conditions may be combined: as when the nutrition of the heart is augmented with the increased action, but still its force is inadequate to its task, and the walls suffer distension at the same time that they are over-nourished. This brief sketch of the pathological causes of hypertrophy and dilatation will render intelligible the circumstances in which they occur. The exciting causes of hypertrophy are habitual muscular exertion of an extreme kind, especially during growth; various diseases of the heart, both functional, increasing its irritability, and structural, exciting its action: these include rheumatism and other inflammatory and febrile diseases; various circumstances impeding the circulation, and taxing the heart with increased labour—such as obesity, tumors, tense pulmonary emphysema, which compress the great vessels, and so forth.

There are a good many anatomical varieties of *hypertrophy* of the heart. It may be general or partial, accompanied by enlargement, or by diminution of the cavities. The size of the natural heart was stated, by Laennec, to be generally about that of the closed fist of the subject. This standard of comparison is more convenient than accurate; and since the more accurate researches of Bouillaud, Bizot, and Clendinning, has been generally superseded by the standard of weight, which averages at 8½ ounces in males, and 7½ in females. The following are about the averages which Bouillaud gives of the chief measurements:—Circumference around the base of the ventricles, 8¾ inches; length, 3⅝ inches; breadth, 2⅝ inches; thickness of walls of left ventricle, little above ½ inch; of right, little under ¼ inch; of the septum, nearly an inch, (this I think too much by one-fourth.) I believe these are pretty accurate measurements for adults, but they must be taken only as averages, subject to fluctuations somewhat above them and below them in varieties of size and age.

In cases of hypertrophy the heart may present various forms and degrees of enlargement, even to three or four times its natural size and weight.* Where the enlargement is considerable, there is always dilatation with the thickening of the walls. Here I would warn the student against the common mistake of using the word hypertrophy as synonymous with thickening. There may be hypertrophy to a great extent without thickening; as where the cavities are much enlarged, and the walls therefore extended. The word hypertrophy means increase of substance, and may be very well applied to the whole heart or to an integral part of it, but not to express the increase in thickness, which is in one direction only. For the same reason I am not well satisfied with the names given by M. Bertin to the varieties of hypertrophy, for they fix the attention too much on the increased nourishment, and too little on the distending influence concerned in their modification. The chief varieties of hypertrophy, are, *simple hypertrophy*, the walls being thickened, but the cavity natural; *hypertrophy with dilatation*, the "eccentric hypertrophy" of Bertin, the walls being either thicker or not thicker than natural. This is the active aneurism of Corvisart: I would, for brevity sake, call it *dilated hypertrophy*. Its converse is *hypertrophy with diminution of the cavity*, which Bertin calls "concentric hypertrophy:" it may as well be termed *contracted hyper-*

* I have met with one which weighed forty ounces; its circumference round the base was fourteen and a half inches; its length from arterial orifices along the septum to the base, eight and a half inches; thickness of left ventricle near the base, one inch and four-tenths; near col. carneæ, one inch and a half.

trophy, which in some measure expresses the cause of the diminution of the cavity. Extreme cases of the latter kind have been met with in persons who have died suddenly, and Cruveilhier and Dr. G. Budd have been led to refer them to tonic contraction of the heart continuing after death; and not to an organic change. I am disposed to agree with these writers.

These several forms of hypertrophy generally affect chiefly one of the compartments of the heart, and none so frequently as the left ventricle; next the right ventricle, and then the auricles. In the latter, dilatation is almost always combined with the hypertrophy. Dilated hypertrophy is, in fact, the most common form in the ventricles also. It is by no means uncommon to see particular parts of a compartment more enlarged than others. Thus the fleshy pillars of the mitral valve, those of the tricuspid valve, the cross muscular stays and network of the interior of the right ventricle, and the musculi pectinati of the right auricle, are frequently developed to an unusual degree. The increase in the pillars of the valves is generally associated with some defect of the valve. I have occasionally seen the thickening of the walls near the base much greater than near the apex; probably from the undue developement of the fibres that encircle this part; rarely the converse is the case. In dilated hypertrophy of the ventricles there is a considerable difference in the shape which it assumes in different cases, the most remarkable varieties being those of elongation and lateral enlargement. Hypertrophy of the left ventricle, with elongated dilatation, is most commonly associated with disease of the aorta or its valves, especially those which permit regurgitation. In disease of the mitral valves or orifice, the dilatation is usually more lateral or globular; but this form is met with also without valvular disease. In the right ventricle, also, the enlargement is in some cases more in the pulmonary, in others more in the inferior or auricular, portion of the ventricle; but I do not know that I can associate these differences with other particular lesions.

The muscular tissue in hypertrophy is not only increased, but it is often more or less changed. In the more sthenic forms, which are associated with active nutrition and a rich state of the blood, it is generally redder and firmer than usual. In leucophlegmatic or cachectic subjects, on the other hand, the increase of substance is often accompanied with duller colour and a laxity and softness which render the texture flabby, and probably cause its being, as it always is in such cases, dilated. In cases of organic disease of long standing, especially with close adhesion of the pericardium, I have repeatedly seen threads or laminæ of a dirty white tissue among the muscular fibres, many of these fibres having also partially lost their colour, and the others being in a flabby state. In other cases, the enlargement of the heart has been in great measure made up of a soft texture containing much fat, like that often found in the pelvis of the kidney. In rare instances, again, whole portions of the ventricles have been found transformed into a dense fibrous tissue. It is obvious that such changes must have an effect on the function of the organ, very different from that arising from simple increase of the muscular fibres. Yet it is not unlikely that they may both originate from inordinate action of the organ, and the modified circulation which it causes through its own vessels, with different conditions of the nutrient fluid.

The pathological effects of hypertrophy will necessarily vary according to its degree, the part which it affects, and the other lesions with which it may be complicated. The commonest and most important form is that affecting the left ventricle, and manifesting its effects on the general circulation and its functions. If the hypertrophy predominate, and be not counteracted by any valvular defect, there will be an increased strength of the arterial pulse, which will commonly be felt most in the arteries nearest to the heart, but it may extend to the whole arterial system. We can readily understand that the increased force of the arterial current may occasion various disorders of function and structure in the several viscera and tissues of the body. In the first place, it may cause dilatation and other changes in the coats of the arteries, especially those that more immediately feel its force, the ascending portion and arch of the aorta. Then as conducted into various tissues and organs, it may excite and disturb their functions, exalt their sensibility, and especially dispose them to inflammation, serous effusion, and hæmorrhage; or aggravate any inflammations, irritations, or hæmorrhages, when they occur. Hence apoplexy and phrenitis, epistaxis, ophthalmia, and other inflammatory affections of the parts of the body, have been traced to this form of disease of the heart. In time, the strong pulse accompanying hypertrophy of the left ventricle may cause an increased or modified deposition of nutriment in the different tissues which it reaches, particularly the parenchyma of viscera. The kidneys afford the best illustration of this, because they receive their blood only from the arterial system. In hypertrophy of any standing they are generally found enlarged, and otherwise diseased, and often presenting the granular albuminous deposit which has been described by Dr. Bright. The lungs and the liver are also very commonly increased in substance; but this must be in many instances partly referred to the obstructed venous circulation which so frequently accompanies hypertrophy of the left ventricle; being another consequence of its cause.* The modification of nutrition especially affects the vascular system, whence arises thickening or ossification of the coats of the small arteries of the brain; and in the rupture of these there appears another mode in which hypertrophy of the heart may lead to apoplexy.

Hypertrophy of the right ventricle is commonly supposed to be a cause of congestion, inflammation, and hæmorrhage, in the pulmonary tissues; but as these effects are seldom observed where there is not also some cause of obstruction to the onward flow of blood through the left side of the heart, it is uncertain what share the hypertrophied right ventricle may have in producing them. In fact, if we remember that its auricular valve is not formed to close completely on an accumulating mass of blood, but permits regurgitation when the ventricle is distended, we can conceive that the pulmonary textures are not likely to suffer from its pulsations, unless their vessels are first distended by some other obstructing cause in or beyond them. In that case the force of the right ventricle must be felt: and when it becomes so much distended as to open its auricular valves, this force is then in part expended backwards into the auricle and veins, causing the venous swelling and pulsation often conspicuous in the jugulars. This venous obstruction, when considerable or permanent, leads to other effects, such as congestions in the portal system and in the sinuses of the brain; whence arise hepatic and gastric disorders, headaches, cerebral oppression, and apoplexy, and especially dropsical effusions of various kinds. These backward effects are the more likely to ensue when the right ventricle is much dilated as well as hypertrophied; whereas the effects on the pulmonary textures, congestion, hæmorrhage, hypertrophy, and excessive bronchial secretion, depend rather on the increased strength, without much enlargement of the ventricle.

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* I have long observed and taught that hypertrophy of the heart commonly occasions hypertrophy as well as change of structure in all the parenchymatous viscera. This has been more satisfactorily proved by Dr. Clendinning in a series of numerical observations of great value.

When the auriculo-ventricular orifice is contracted, we occasionally find dilated hypertrophy of the right ventricle combined with contracted hypertrophy of the left. It has puzzled pathologists to account for this hypertrophy of the left ventricle, when its task must be diminished by its receiving less blood to propel from the left auricle. Dr. Hope has attempted to explain it by the left ventricle feeling the obstacle at its own auricular orifice through the whole course of the arteries, capillaries, veins, the right side of the heart, and the pulmonary vessels; a roundabout explanation truly, and one that supposes a degree of distension of all these parts that is very rarely seen. Then Dr. Copland urges this case as an argument for his favourite notion of an active expansion of the ventricles, which I cannot admit to be warranted by any physiological analogies. But how very simple is the true cause of this hypertrophy, if I understand it aright! Why should not the excitement of the whole heart, dependent on the distension of all its other cavities, produce increased action, and eventually increased growth, of the left ventricle also? Is it possible that the same fibres which encircle both ventricles can be excited in one, and not in the other? No, surely; the left ventricle, naturally the strongest and most active, is thus excited by sympathy or continuity of irritation; and when its walls become increased under this influence, the cavity must be contracted from the smallness of its contents.

I am not aware of any remarkable pathological consequence of hypertrophy or dilatation of the auricles: they are to be considered as effects more than as causes. The strength of their contraction is too trifling to produce any other effect than that of stimulating more powerfully their ventricles, which are apparently excited to contraction by their impulse. This influence is perhaps reciprocal in the case of the right auricle and ventricle; and the consecutive pulsations of the two propagated backwards are sometimes seen in a remarkable double pulse in the jugular veins. The dilatation of the right auricle is sometimes enormous. I have seen it large enough to contain an ordinary-sized heart.

From the general pathological effects of hypertrophy of the heart we may infer the prevailing character of its symptoms. A forcible, at the same time often not a very effective, state of the circulation, with its phenomena, palpitation and throbbing, occasional flushing, and a strong hard pulse, manifesting its effects on particular organs, as by giddiness, palsy, or apoplexy in the brain; dyspnœa, asthma, with thin mucous secretion, or occasionally hæmorrhage in the pulmonary tissues; gastritic dyspepsia, gastrodynia, sickness, discoloured alvine secretions, and other symptoms of disorder in the stomach, liver, and bowels; albuminous, high-coloured, and often scanty urine, with accompanying gouty or dropsical symptoms, referable to disordered functions of the kidneys: more or fewer of these may be variously grouped together in different cases, as frequent effects or symptoms of the more sthenic forms of hypertrophy of the heart. Some of these secondary effects may occur and be the subjects of complaint, when those of the immediate action of the organ, the cause of them all, are not sufficiently marked to attract attention. When dilatation is joined with it, there will be more weakness of the circulation, with consequent torpidity of the functions of the extreme vessels, manifesting itself in venous or visceral congestions, œdema, and coldness of the extremities, pains in the back and limbs; and the incapacity of the organ will be greater; whence a pulse unequal in force and often irregular in rhythm, severe attacks of palpitation and dyspnœa may then present themselves, and the disease is more manifestly one of the heart itself.

The physical signs of hypertrophy are intelligible and characteristic. The increased mass of the muscular fibres renders their act of contraction stronger than usual; hence the impulse is also strong. But the character of this impulse, as well as the sound which attends the contractions, will depend on the form which the hypertrophy has assumed. When it is a simple thickening of the walls without increase or diminution of the cavity, the impulse will be gradual and heaving as well as strong, both because thick muscles cannot contract so simply or abruptly as those that are thin, and because the enlarged size of the heart brings more of it in successive contact with the ribs. So, likewise, the first or systolic sound will be prolonged, but duller than usual, because the sounding transition of thick walls, from loose to tight, is less extensive, abrupt, and instantaneous, than when they are thin. I formerly illustrated this by the different sounds produced by suddenly stretching fabrics of different thickness, such as linen and sackcloth, or thin stuff and thick baize. With contracted hypertrophy, in which the cavity is smaller, and the walls therefore have less extent of motion, the diminution and dulness of the first sound in the region of the heart is still more remarkable; it may be quite destitute of the flap usually heard, whilst the impulse is remarkably heaving, as if the heart swelled against the ribs at each contraction. It often happens that the sound is louder at the top of the sternum and in the carotids, than in the region of the heart. The second sound, as heard at the mid-sternum, may be as loud as usual. When, on the other hand, the hypertrophy is dilated, there are better conditions for generating sound; the walls and valves, being loose and flabby, pass with greater abruptness into the tense state of contraction, and yield a louder sound; whilst the impulse, although strong, is more abrupt, and in cases of extensive disease is followed by a motion of collapse, from the sudden falling back of the large heart into a state of passive looseness at the moment of diastole. Both sound and impulse will then be extended over a wider space than is natural; and when the disease is considerable, and unattended with an emphysematous state of the lung, there will be also dulness on percussion, more or less extensive according as the enlarged heart is in contact with the thoracic walls. The situation of this dulness, and of the impulse, will vary according to the form of the dilated hypertrophy and other circumstances which affect the position of the organ. In elongated enlargement the impulse is generally felt below its usual spot, between the fifth and sixth ribs, down to the seventh or eighth; and I have even felt it in the abdomen, below the margins of the ribs. The dulness reaches from that part upwards to the sternum. But constant, or even occasional distension of the abdomen, by any cause, which prevents the descent of the diaphragm, will make the enlarged heart take another position, by which its apex extends further to the left, and the dulness on percussion reaches from that point to the sternum, occupying the whole infra-mammary, and perhaps part of the mammary region. The same position of the heart, thus more horizontal than natural, may, perhaps, also be caused, as Dr. Hope has pointed out, by adhesions of the pericardium, which prevent the organ from enlarging downwards. Enlargement of the liver, distensions of the stomach or colon, and dropsical effusions in the abdomen, are the most frequent causes of this lateral direction which enlargement of the heart takes. Similar causes may also determine the displacement to be outwards, against the thoracic walls, occasioning them to project to the left of the sternum, in the manner described by Bouillaud and Louis. This is most remarkable in children, and in young persons with narrow chests. When the enlargement is more transverse or globular, the dulness on percussion, and the impulse, are higher, independently of the position of the diaphragm; and this is particularly the case when the right ventricle is also enlarged.

I have known the dulness on percussion and impulse to reach over two-thirds of the left front of the chest, and as high as the third rib. We can understand why, under these circumstances, the impulse should be of a different kind as well as more diffused than usual, being produced by the swelling *body* of the ventricle rather than by the striking apex, and is therefore also felt higher instead of lower in the chest.

I have said that naturally the left ventricle touches the walls of the chest only at the apex, and that the impulse felt on full expiration at the sternum is that of the right ventricle. But if the left ventricle be much enlarged, and the right not, its impulse may be felt as far as the sternum, the impulse and sound of the right ventricle being at the sternum only, or transferred to the right of it and at the epigastrium. Again, if the right ventricle be also enlarged, it will keep the left ventricle to the left, and from the walls of the chest, so that its stronger impulse may be but little felt, nearly the whole front being occupied by the dilated right ventricle. Hence the reason why much enlarged hearts are often not felt beating with the force that might be expected from the thickness of the walls of their left ventricle; hence, too, the rules given by Laennec and others, to find the signs of each ventricle at particular spots, fail in many cases. The position of the whole heart, and of its compartments, is changeable, and when altered by disease it may be so different as to be entirely shifted from the common *landmarks*. It is rather by relative than by absolute rules that we can rightly judge here. For instance, if an hypertrophied left ventricle encroaches on the region of the right, we may know still that it is the left, by finding the different kind of sound and impulse to the right of it, and no other kind to the left. When the heart is displaced to the left, the signs of the right ventricle are those in the lower as well as right part of the space in which the sounds and impulse are felt most, those of the left ventricle being above and to the left.

To a certain extent we may also judge the strength of the left ventricle by the loudness of the sounds and the strength of the pulse at the top of the sternum and in the carotid arteries, when they are not exaggerated by dilatation of the aorta. Hypertrophy of the right ventricle could not cause these signs, whatever increase of sound or impulse it may produce in the region of the heart itself. Again, when the pulse is strong, and the sounds loud at the top of the sternum and in the carotids, although there be no strong impulse to the left of the sternum, there is probably hypertrophy of the left ventricle, but it is displaced from the walls of the chest by a dilated right ventricle, expends its force on the more yielding tissue of the lung, and in slender persons may be actually felt in the left back. On the other hand, tumours, or a consolidated lung, which push forward the heart or prevent its receding from the walls into the spongy lung, must greatly exaggerate the impulse of the heart, and may give the impression of extensive hypertrophy where little or none exists. I have seen such effects from aneurism of the descending aorta, from tuberculous or inflammatory consolidation, and tense emphysema of the inferior lobe of the lung, and from extensive effusion in the right pleura. For analogous reasons hypertrophy of the heart is more perceptible and distressing in persons with narrow or distorted chest than in others. Many individuals with broad deep chests have large hearts that give them little or no trouble, but would cause distressing symptoms in those of a slender make.

When, instead of increase of the muscular tissue, the heart is enlarged by the deposition of fat around it, the sound is obtuse as in hypertrophy; but the arterial pulse and cardiac impulse are weakened also, and the dulness is more at the sternum, or middle portion of the chest.

Dilatation of the Heart.

Dilatation of the heart is in many respects the converse of hypertrophy. When the heart is incapable of sufficiently expelling its contents, whether in consequence of obstruction in the vessels from it—of regurgitation into it through imperfect valves—of want of power, of irritability, or of both, it becomes distended, and in time permanently dilated. The circumstances which determine these conditions, that is, the occasional or exciting causes of dilatation, are weakening influences in general, such as those of long-continued fevers, cachectic states of the system, with bad blood; and causes which weaken the heart in particular, such as continued inflammation, obstruction of the coronary vessels, and such circumstances as injure the nutrition of the organ.

The anatomical characters of dilatation are not only the thinning and extension of the walls of the affected compartment, but also generally a paler and more flaccid condition of their muscular fibres. In some parts, particularly of the auricles, and at the apex of the left ventricle, the attenuation of the walls has sometimes proceeded so far, that the pericardium and endocardium are in contact, and these are occasionally thickened by opaque deposit, as if to strengthen them at these parts. The right ventricle and the left auricle are the most common subjects of simple dilatation. In the other compartments it is occasionally met with, but more generally combined with some degree of hypertrophy; so that although the walls be thinner than natural, their greater extent gives them an increase of substance. The right auricle and ventricle are sometimes dilated to an enormous extent, with thinning of their walls, but still increase of substance; and this condition is generally found to be associated with disease of the mitral valve. Dilatation of the ventricles is commonly in all directions, rendering the cavities globular; but it is occasionally partial, the walls being distended into a pouch or aneurism, which in rare instances attains a considerable size, and may end in rupture.* With the dilated condition of the walls there may appear various other traces of disease in the lining membrane and valves; such as opacity, thickening, and roughness. They are most seen near the valves and orifices, and are evidently the result of an inflammatory process, which may have been either the cause or the effect of the dilatation. The orifices and their valves are commonly somewhat dilated, as well as the other walls, so that they may still maintain a sufficient proportion to perform their office. The dilatation of the semilunar valves is considerable, and renders them so thin that the fibrous threads can be seen forming an irregular network in them. Their thinning sometimes amounts to perforation, especially at the margins which apply to each other, and then this fibrous network may be the only part left. The tricuspid valve is seldom expanded in proportion to its orifice, which almost always partakes of the dilatation of its ventricle; hence there is free regurgitation through this orifice.

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* A very complete memoir on Partial Dilatations and Aneurisms of the Heart, by my friend Mr. Thurnam, has lately appeared in the Medico-Chirurgical Transactions.

The pathological effects and symptoms of a dilated heart are such as we have already described as those of irregular and defective action. Having here become an effect of permanent change of structure, they are necessarily more enduring than where they are merely functional; and there are added to them others, arising from this more permanent influence on the other structures and functions of the body. When the left ventricle is dilated, its propulsive power is impaired; there may be faintings, alternated with palpitation and other irregularity; the whole systemic circulation is languid, and causes the symptoms of muscular debility, coldness of the extremities, defective and disordered secretions, imperfect nutrition, and eventually œdema of distant parts. When the change is chiefly in the right ventricle, the symptoms are those of impeded venous circulation; lividity of parts naturally red; anasarca, often not confined to the lower extremities, perhaps with ascites and general congestion of the abdominal viscera. To these are often added the symptoms of pulmonary congestion, with its distressing concomitants, asthma, cough, profuse expectoration, and hæmoptysis, induced by the same cause which led to the dilatation of the right ventricle—disease in the mitral orifice. The complication of dilatation with valvular disease is particularly unfavourable, for it constitutes a double defect in the power of the organ, whilst its irritability may continually excite it to fruitless and most harassing efforts of palpitation, which may terminate life by ending in syncope, or wear it out in a more gradual manner.

The physical signs of dilatation were well described by Laennec; they are the converse of those of hypertrophy, and are equally intelligible and characteristic. Extended into a thin layer of muscle, the contractions of the walls are simple, abrupt and quick; and thus, although they have little power, they are well calculated to produce a loud sound and slight brisk impulse; and these are diffused over a wide space, proportioned to the extent of the dilatation and its proximity to the walls of the chest; but the sound can have little duration, and the impulse little strength; and even under the influence of palpitation the pulsations do not raise the walls, as an hypertrophied or even a healthy heart does when excited. The first sound of a dilated heart so closely resembles the second, that it is not easy to distinguish between them without placing the hand at the same time on the region of the heart, or on one of the carotids, to find which sound corresponds with the pulse. I should express this sound by the word *lup-tup*, instead of the *lubb-dup* of the healthy heart. The sound on percussion will be also dull in the region of the heart, in some proportion to the extent of the disease; more extensively so than is usual in simple hypertrophy, but less than in dilated hypertrophy, in which the greater size to which the organ attains, and its flaccidity, which exists as in dilatation, brings a larger surface in apposition to the walls of the chest. To a certain degree we may, by the position of these signs, determine which compartment of the heart is most dilated, those of the left ventricle being, as Laennec pointed out, situated between the cartilages of the fifth and seventh ribs, and those of the right at the lower half of the sternum, and in the epigastrium. A dilated right ventricle, however, sometimes extends far to the left of the sternum, carrying the left ventricle behind and above it away from the walls of the chest, which, as in dilated hypertrophy, may prevent the impulse and sounds of the latter from being readily distinguished from those of the right. Under these circumstances a complete expiration, and leaning forwards to the left, will sometimes bring the heart into more complete contact with the walls of the chest, and give the stronger impulse and duller sound somewhere under the left breast. On the other hand, when the left ventricle is dilated, the impulse is felt lower than usual; but it does not displace materially the position of the right ventricle, which may be heard and felt at the sternum. In consequence of their greater loudness, and the large size of the body in which they are produced, the sounds are heard over a greater extent of the chest than usual; but I cannot agree with Laennec, that this extent is so perceptibly proportioned to the degree of the dilatation as to be an exact measure of it. The transmission of the sound through the lungs may be so much modified by even temporary conditions of the lungs (and much more so by consolidation, hypertrophy, or emphysema), that the distinctness of the sounds in different parts of the chest depends as much on these organs as on the intensity of the sounds themselves.

Extreme dilatation of the auricle, particularly the right, may cause dulness on the percussion at the middle of the sternum, and on either side of it; in fact, it only adds to the extent of the dulness produced by the enlargement of the ventricle which is conjoined with it. Laennec taught that dilatation of the auricles increased the loudness of the second sound; and although his view of the cause of this is totally erroneous, I think I can from experience confirm the fact, that the second sound has been sometimes unusually distinct at the middle of the sternum and on either side of it, in cases in which examination after death showed one or both of the auricles to be dilated. The second sound is produced by the flapping tension of both sets of semilunar valves; and although the dilated auricles which lie close to these valves do not increase the sound, they transmit it to the front wall of the chest better than the spongy tissue of the lung, which generally intervenes, could do. I once saw a double pulsation between the third and fourth ribs, close to the sternum, which I was led to ascribe to a greatly dilated right auricle. The same double pulse was in the jugular veins; and I believe, with Laennec, very commonly attends extreme dilatation of the two right compartments of the heart, there being, in such a case, pretty free regurgitation through the auriculo-ventricular orifice.

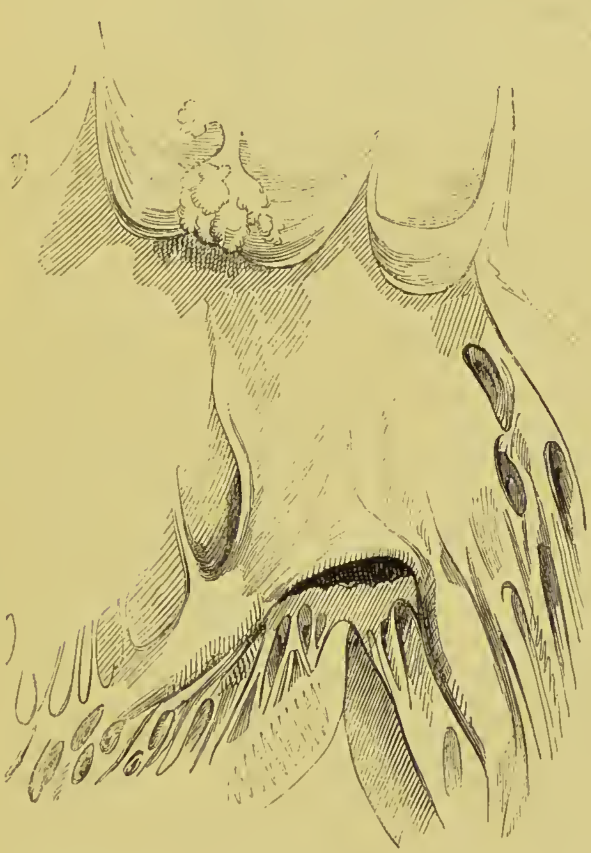
Diseases of the Orifices and Valves of the Heart.

We have seen a little of the effects which result from diseases of the fibres which move the blood; we have now to attend to the chief lesions of that mechanism by which this motion is directed and conducted—the orifices, with their valves and great arteries.

Diseases of the orifices and their valves are of very considerable variety, but those that interfere with the office of the parts which they affect may be reduced to two classes—those that more or less *obstruct* the current of the blood in its proper channel, and those that occasion it to take a *reversed* direction. The former, for brevity, I term *obstructive*, the latter *regurgitant* lesions.

The most common change in the valves is *thickening*, which presents itself chiefly under two forms:—1. A *softer* kind of *thickening*, in which the valves retain much of their pliability; 2. An *opaque thickening*, with more or less *induration*, so that the valves become less flexible and mobile than usual. The first occasionally affects the semilunar valves, but rarely the auricular. It appears to be the product of inflammation chiefly affecting the serous membranes of the valves, and is produced by organized lymph between their layers, or upon their exterior, from which it often may be separated. This deposit is generally seen less at the margins than at the middle and attached parts of the semilunar valves, chiefly on their ventricular sides, and occasionally forming a bond of *adhesion* between two adjoining valves, which are glued together as far as the corpora arantii, as seen in fig. 1. This constitutes a form of slight obstructive disease of the arterial orifice. I have seen the same deposit on the layers and cords of the auricular valves, causing them to adhere, and forming a false membrane on their auricular surface.

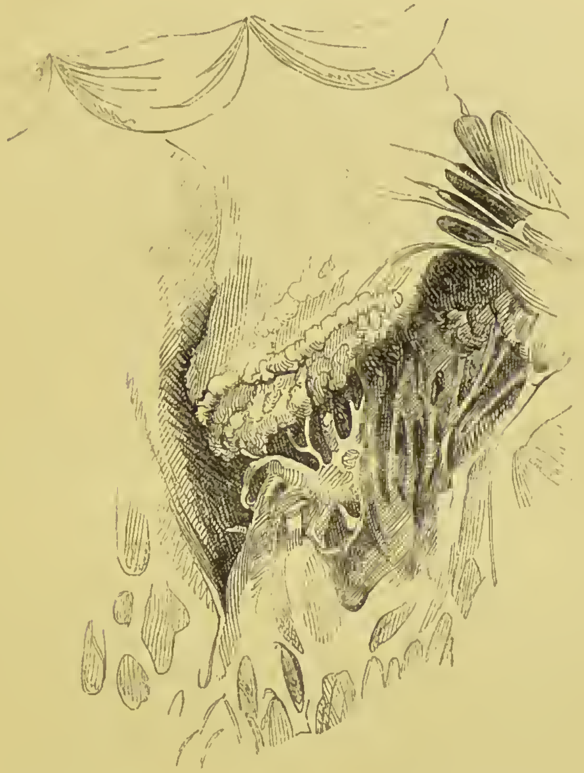
Fig. 1.



Adhesion with fibro-osseous deposit between two aortic valves—agglutination of tendons of mitral valve, and contraction of its orifice. Ph. signs.—Blowing mur. with 1st sound heard at apex and sternum—2d sound distinct at sternum. Subject, "asthmatic" ever since six months of rheumatism, 30 years before death.

The *opaque tough thickening* of the valves is the most common, and is frequently combined with the former. When occurring simply, there is a smoothness of the exterior, which, with the character of the thickening material, seems to indicate that the deposit is chiefly between the serous layers of the valves, and probably arises from disease in the fibrous tissue, that forms the strengthening web of the valves. This thickening commonly affects the semilunar valves to their very margins, and is sometimes seen extending into the ventricular lining, and perhaps forming there slight ridges where the bands of fibrous tissue cross the muscular walls. With the laminæ of the auricular valves it commonly involves their orifices and part of the auricular lining, and extending into the tendinous cords, which are irregularly thick and knotty, and their fleshy columns are likewise occasionally changed into fibrous cords. With this more rigid thickening there is often combined a *contraction* or *elongation* of some parts, or both, affecting different parts ; and it is such changes of proportion that so commonly injure the proper action of the valves and orifices. Mere thickening, unless it be attended with great stiffness, will not materially injure the office of the valves; but changes in form and proportions necessarily must. The annexed drawings illustrate some of these changes. In those affecting the mitral valve, the shortening of a few of the tendinous cords throws the rest into a loose state, in which they cannot draw the membranes of the valve smooth on each other ; hence they form folds and chinks, through which the blood must regurgitate at each systole. In other instances portions of the valve and their cords are elongated and enlarged, so that they may be forced backwards towards the orifice, and, by keeping the valve partially open, also occasion regurgitation. (Fig. 2.) In other cases, again, the two laminæ of the valve and their cords are thickened, contracted, and adherent, forming a sort of narrow funnel-shaped tube instead of a valve, both retarding the flow of blood from the auricle into the ventricle, and never closing against its reflux into the auricle. (See fig. 1.) This forms both obstructive and regurgitant disease of the mitral orifice. In this manner the left auricular orifice is sometimes so contracted as not to admit a little finger; naturally it admits with ease two full-sized fingers. In the aortic valves we sometimes see one of them contracted at its free margin, so that it cannot meet its fellows, but leaves a chink through which the blood regurgitates with a murmur into the ventricle at each diastole.

Fig. 2.



Rupture of mitral tendons. Warty and fibrinous concretions.
Ph. signs.—Loud blowing at apex.

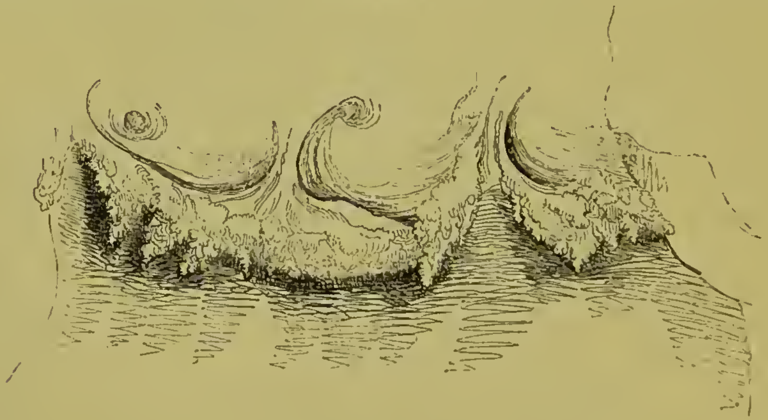
Again, an uncommon elongation of the free margin or attachment of one or two of the semilunar valves, so that these margins, instead of being closed against their fellows, become retroverted under them, constitutes another form of regurgitant disease of the semilunar valves.

To the thickening of the valves and orifices there are frequently added great induration and even ossification; and this alone may constitute disease, generally of the obstructive kind. As such, it often affects the aortic orifice, particularly at the attached portions of the valves, and at the curves of the artery, which form the boundary of the little recesses or sinuses into which they retire. If, in either of these situations, a rigid portion project into the current as it passes, it constitutes obstructive disease, and may cause a constant murmur with each pulse; or if the whole ring be rigid and unyielding, it may only, under the circumstances of greatly increased action, constitute a constriction, the vibrating resistance of which, opposed to the current, may give a murmur.

The lesions which I have been describing are the most common forms of diseases of the valves; they are mostly of a chronic character, and in the greatest number of instances originate in rheumatic endocarditis. Their effects on the circulation and on other functions have been generally proportioned to their degree and character, the rapidity of their production, and to their complication with hypertrophy and dilatation, or other disease: hence they may vary infinitely; they may exist and produce cognizable signs, without obviously deranging the heart's action, or in an overt direct manner injuring the health; and they may cause the most distressing and dangerous symptoms, and sooner or later prove fatal.

There is another kind of thickening to which the valves are subject, accompanied with *softening*, *ulceration*, and, often *rupture*. This is fortunately not very common; for it is a terrible disease, destroying the valves, chiefly the aortic, in the course of a few weeks; and soon after proving fatal. This occasionally supervenes on older disease which has originated in rheumatism; but I have in several instances known it to arise independently of rheumatism, perhaps after a severe cold, or violent strain, especially in persons who have been addicted to spirits. The ruptured or ulcerated portions of the valves are found loaded with ragged, soft, fragile vegetations, more or less tinged with blood; and these are also sometimes seen adhering to adjacent parts where the endocardium is entire. The remaining parts of the valves are much thickened, and opaque yellowish white, with a pink hue; and pink patches

Fig. 3.



Aortic valves broken down, thickened, and fringed with vegetations.

Ph. sign, deep-toned double murmur at sternum and in carotids.—In a drunkard, fatal in six weeks.

are often seen in the aorta, with atheromatous thickening. I think that these changes may be viewed as the effects of acute inflammation affecting all the tissues of the valves. In Fig. 3, all three aortic valves have been completely broken up, and their torn margin, fringed with thick vegetations, hangs down into the ventricle. In other cases I have seen smaller perforations, probably ulceration, both in the middle of the aortic and in the mitral valves; always fringed with vegetations. The smooth perforations, so common at the free margin of the semilunar valves, are of a different character, and not dependent on the same cause. I believe that in time the vegetations just described may become organized, forming the cartilaginous or fibrous little bodies which have been called warty excrescences, in which osseous matter is often found, and which generally have more recent vegetations attached to them.

Lastly, we have another class of valvular diseases that have been very commonly overlooked. I mean *atrophy* or *wasting* of the valves, by which their membranous portions may become *shortened* or *perforated*, and the tendinous cords withered and absorbed away. The semilunar valves, both aortic and pulmonary, present these in the most obvious manner, in the oval perforations at their free margins. The perforations are oval or rounded, with their edges quite smooth and thin, as is also the whole valve. In fact, although there may be partial deposits of false membrane on their ventricular surface, the valves are most commonly very thin and flaccid when they present these perforations; and in other parts there may be seen still thinner spots that are separations of the fibrous web, and are all but through the serous membrane also. Now, so long as these perforations are confined to so much of the margin of the valve as closes against that of its fellow, they may not produce regurgitation; and this is very commonly the case. In the mitral valve, the wasting usually affects the posterior portion, the membrane of which is often annihilated by it, the cords being inserted directly into the auricular ring. The anterior lamina is also occasionally found much shortened, and without those fine thin expansions of membrane which commonly unite the cords to each other, below their insertion into the thicker part of the valve. It is pretty plain, that with this state of the valve, if there be not habitual regurgitation, inordinate action of the heart, or slightly disturbing circumstances, may induce it, especially if there be at the same time dilatation of the orifice. I have known a murmur produced by flatulent distension of the stomach, and by certain postures, which I have been inclined, for reasons to be explained afterwards, to refer to this kind of imperfection of the mitral valve. I have found these atrophied conditions of the valves in cases where there was no trace of previous inflammatory affection of the heart.

With these several forms of valvular disease, especially those of an inflammatory origin, there are commonly combined various forms and degrees of hypertrophy and dilatation; the production of which may be more or less ascribed to, and explained by the nature of the valvular disease.* Thus hypertrophy with dilatation commonly affects a ventricle the orifices of which are partially obstructed, or permit regurgitation, either of which conditions tend unduly to irritate and distend it; and as the efforts of the ventricle continue to be powerful or not, the hypertrophy or the dilatation will prevail.

The valves of the left side of the heart are far more commonly diseased than those of the right. Yet occasionally the latter do present the same changes as the left valves, but it is very rare that they are alone affected, or in a much greater degree than the left. Nor can we wonder at this, when we consider the function, structure, and relations of the left side of the heart as compared with the right. Its function requires that it should be endowed with much greater strength, that it may propel the blood through the great circulation; and this greater strength exposes its parts to more violence from its own movements. Then, to bear this violence, its valves and orifices are furnished more abundantly with a strengthening fibrous tissue; yet this very tissue is obnoxious to inflammatory affections, which tend peculiarly to alter it. A tissue which protects by its strength cannot be endowed with high vitality; as its vitality is low, so is its natural reparatory power slow, and the hastening of this process by inflammation changes the nature and strength of the material. Hence there are deposited no longer the fine, even transparent, capillary fibres, which only glisten into view with a silvery whiteness when they are bundled together in numbers, but an opaque, yellowish white, thick, tough material, partly fibrous and partly amorphous, possessing neither the strength nor the delicate flexibility of the original texture, and consequently injuring the apparatus by its bulk, its stiffness, or its liability to extension or contraction, laceration or rupture. Then the very perfectness of structure, that gives to the left ventricle a superiority in point of strength over the right, exposes it more to the effects of violence or excessive action. When the right ventricle becomes distended, its auricular valve opens and permits a partial reflux; but the mitral valve of the left ventricle is made to close perfectly, and to bear the whole strain of the muscular fibres contracting on the blood. Again, the pulmonary valves are exposed to pressure from the tension of the pulmonary vessels also, which are exposed to little pressure but that of the expiratory forces; but the aortic valves receive the strain of the great arterial system, liable as this is to violent increase from the pressure of the muscles of the body, and of any sudden blow or impression on regions containing its larger branches. We cannot wonder, then, that the valves of the left ventricle should be more frequently and more extensively diseased than those of the right. The atrophous form of disease, however, which produces the thinning and oval perforations in the semilunar valves, and wasting of the membranous parts and tendons of the auricular, is commonly met with in either compartment of the heart. It was on the right side that I have seen it attain the greatest degree, having reduced the membranes of the tricuspid to mere fringes, and the margins of the pulmonary valves to a network of threads. But even such disease on this side of the heart produces less prominent symptoms than slighter lesions on the other.

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* I can by no means subscribe to the opinion of Dr. Clendinning, that valvular lesions are secondary in importance, or in time, to the morbid conditions of the walls which accompany them. I suspect that we differ as to what is and what is not morbid in the state of the valves. This question must be reserved for another work.

We shall now consider the *pathological history* of diseases of the valves; and as a distinction between them can scarcely be made without a study of their *physical signs*, we will begin with this subject, which will supply us with further knowledge of their more essential pathology. I have before had occasion to mention that the phenomena called *murmurs* are often produced by the modifications which valvular disease occasions in the current of the blood. In order that they be produced, there must be the requisite elements, sufficient force in the current, and such a modification as may constitute a vibrating resistance to it. We can understand that valvular disease may be present without forming these elements, and therefore without any attendant murmur, as when the current is too languid, or the resistance too loose or yielding to cause vibrations; but, in by far the greater proportion of cases, valvular disease does cause murmurs, either constantly, or under the circumstance of the current increased by exertion or other cause of excitement.

I hope that the reader now comprehends the principles of the production of sound too well to fall into the common error of supposing that the amount of a valvular lesion is indicated by the *loudness* or extraordinary character of the murmur. The loudest and most singular murmurs are the whistling, cooing, or musical, yet they are occasionally produced by comparatively slight lesions. The loudest musical murmur that I ever heard was produced by the retroversion of only the margin of one of the aortic valves, the rest being in a pretty capable condition. The murmur was a loud cooing with the second sound; and the patient dying of fever, I had the opportunity of verifying the diagnosis which I had made of the disease. Again, in the case of Fig. 3, where all the aortic valves were entirely broken down by disease, the murmur, which was double, was obscure and distant.

The *quality* of the murmur is a better index to the character of the lesion, but it is by no means a sure one. Uniform and shrill murmurs, whether blowing or musical, generally indicate slighter lesions than the rough or grating and deep-toned, because they are commonly produced where there is no great looseness or irregularity of the aperture through which the current generates the sound; they are most frequently caused by regurgitation through small smooth chinks between the mitral or the aortic valves. The rough grating or sawing sounds generally indicate more extensive disease, not being produced (as it has been asserted by others, and stated in my former edition on their authority) necessarily by indurated or osseous irregularities, but by a larger current passing over a body which offers to it an irregularly vibrating resistance. Such murmurs most commonly occur in the aortic orifice.

The more distinguishing circumstances in regard to murmurs are, however, their relation to the natural sounds or motions of the heart, and the points at which they can be best heard. Laennec and Bertin paid some attention to these points, but without obtaining any general results. I believe that Dr. Elliotson was the first who attempted a diagnosis between different valvular lesions by the apparent situation of their murmurs; but neither this attempt, nor that subsequently made by Dr. Hope, was founded on correct acoustic principles, and the means of distinction announced by them have failed when tried by others. The latter asserted that the disease of a particular valve may be known by the murmur being heard best at a spot on the walls of the chest nearest to that valve, and by its being of a higher or a lower key, according to the proximity or remoteness of the valve from the walls of the chest. But the position of the different valves is too close for such a distinction: all four sets are comprised within a space of a couple of inches, being placed, in regard to the front of the chest, one behind another rather than side by side. Dr. Joy has proved that the end of a common stethoscope may cover the greater part of all of them.* Then the lung intervenes between them and the front wall of the chest, and must obscure and confound the sounds from beyond it; so that it is impossible in so small a space to discriminate between the murmurs generated at the different orifices. But there are principles of diagnosis in these murmurs, and if they be carefully and rationally studied, they will generally enable us to distinguish, as far as is useful, between the different kinds of valvular lesion. They are nearly such as I gave them in the last edition of this work; and I have since been enabled to confirm and correct them by the examination of very many cases before and after death.

In judging of the seat and cause of a cardiac murmur, we have to attend chiefly to the *period* of the heart's motions at which it occurs, and the *manner* in which it is *transmitted* to the surface. A murmur accompanying only the first sound or impulse is necessarily caused by a current of blood *from* a ventricle; one accompanying, following, or replacing the second sound, must arise from a current *into* a ventricle. This simplifies the matter; but there are still two ventricles and two orifices to each; and how are the murmurs of these respectively to be distinguished? Not by the different positions of these orifices certainly; they are too close together for that; but *by the different directions in which the sonorous currents spread the sounds*, and *the different manner in which they are transmitted to the walls of the chest.* Thus murmurs generated at the origin of the arteries will generally be more or less transmitted in the direction of the current along these arteries; and those produced in the auricular orifices will be conducted both by the current into the ~~auricles~~ tricles, and by the tightened cords and fleshy columns to the apex of the heart, which is generally more or less in contact with the ribs. So far for the general principles: now let us consider briefly the special diagnosis of particular lesions.

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* As Dr. Hope has, in his last edition (1839), in great measure abandoned these rules of diagnosis, and adopted mine without acknowledgment, I must refer to dates to establish my originality. In 1835 were published, at the same time, Dr. Hope's "second edition" and my third edition: the former containing the erroneous rules above noticed, which he has now abandoned; the latter, my rules, which have for the most part held good ever since. In March, 1836, I published in the Med. Gaz. cases illustrating the successful application of these rules, verified by dissection. These cases and the diagnoses were well known to some of the physicians and pupils of St. George's Hospital. Dr. Hope's *new* rules first appeared in Aug. 1838, and more fully in his third edition in 1839. These rules are, in the most essential points, the same with those published by me more than three years before. The chief point of difference is in his still making the *pitch*, or *key*, of the murmur a distinction between a superficial and a deep-seated murmur. Now in this he is quite in error, as many of his own cases would prove. The key of a murmur may be higher in the aortic than in the pulmonary, and in the mitral than in the aortic orifice. Distance affects *timbre*, or quality and intensity of sound, but not its key. This is taught in all works on acoustics.

Obstructive disease of the *aortic orifice* (by *obstructive* I do not mean that the actual impediment is always much) is generally attended by a murmur with the first sound and impulse, heard in the region of the heart, along the upper half of the sternum, and in the right or both carotid arteries. The point where it is heard loudest will vary according to the position of the heart with regard to the lungs and walls of the chest. If it lie deep, and be well covered with spongy lung, the murmur may be louder at the apex and in the right carotid than at any intermediate point, because the dense heart and the distended arteries convey the sound better than even a much shorter length of pulmonary tissue. In chests less deep, and especially where enlargement of the heart brings its base nearer to the sternum, the sound is loudest about the middle of this bone, or a little on either side of it; but still the more distinguishing character is, that it is heard above, in the direction of the innominata and carotid arteries, where sounds from the other valves can scarcely reach. The murmur is commonly like grating or sawing, and varies much in length and loudness in different cases. In some it lasts the whole period from the commencement of the systole to the occurrence of the second sound: in others it is a mere roughness or whizzing accompanying the natural sound, which may be pronounced *trrhub-dup* or *djub-dup.* It is very often accompanied by regurgitant disease of the aortic valves; and when this is not the case, the second sound (the *dup*) is frequently less clear than usual, which implies that the valves, from thickening, do not act freely.

Regurgitant disease of the *aortic orifice* produces a murmur with or instead of the second sound, by the sonorous gush of blood from the artery back into the ventricle. The place of greatest distinctness of this sound will also vary, as the former, according to the position of the heart and lungs: it is generally heard best at or near the middle of the sternum; but its more characteristic feature is, that it obscures or supplants the second sound or flap at the top of the sternum and in the carotid arteries, which is usually very distinct there. The second sound of the right or pulmonary valves can often still be heard about the left margin of the mid sternum. This murmur is seldom so rough or grating as that of obstructive disease; for the current refluent from the artery is not so large or strong. It may, however, contrary to the statement of Dr. Hope, be very loud, and even musical, as in the case which I before mentioned; and if the valves cannot close at any period of the diastole, it may even last from pulse to pulse. More commonly, however, it dies away before the next pulse; and, in slighter degrees of regurgitant disease, is very short, preceding and not supplanting the second sound. This is when the valves, before they are quite distended by the full recoil on them of the arterial column, permit a little jet of blood to grate between their margins, and make the second sound like *trrht* or *tzzt*, instead of *dup*. A little vegetation, or slight deposit of rough lymph on their margins, may cause this: I have often met with it both as a temporary and as a permanent phenomenon, when there was not much derangement of the heart's action.

Combined obstructive and regurgitant lesions of the *aortic orifice* produce the double, or to-and-fro sawing murmur, which is the succession of the forward and backward gushes of blood. They are by no means uncommon; in fact, the murmur, instead of the second sound, indicating regurgitation, seldom exists without a murmur with the first also, indicating partial obstruction. These lesions, when at all considerable, are accompanied by dilated hypertrophy of the left ventricle, which often takes an elongated form, bringing its impulse lower and more to the left than usual. When the regurgitation is very free, it increases the force of the diastolic collapse so much, that I have known it resemble a second impulse. It also not unfrequently stimulates the ventricle to a second contraction, constituting a reduplication of the pulse: the rhythm otherwise is not commonly much affected. But the pulse generally possesses a character that is remarkable. When the lesion is obstructive only, the pulse is generally hard and jarring; but when it is considerably regurgitant also, each pulse, although momentarily hard and full, immediately recedes, which gives it a jerking or thrilling character. The same circumstance makes the pulsation of all the arteries *visible*, and sometimes *locomotive*, —a sign of lesions of the aortic valves first pointed out by Dr. Corrigan. I have seen it so extensive in an old man, that many arteries could be seen like worms under the skin, wriggling into tortuous lines at each pulse. The cause of this phenomenon is sufficiently intelligible. It proceeds from a defective equality of tension of the arterial coats. These vessels are distended at each pulse, and emptied the instant after, the aortic valves not, as usual, maintaining their tension; and if the coats of the arteries are defective in lateral elasticity, as in old people, they may admit each jerking jet of blood only by being elongated into a tortuous line. In its extreme degrees, and especially when existing in all states of the circulation, I think that this visible or moving pulsation of the arteries is pathognomonic of regurgitant disease of the aortic valves; but to a slight extent it may be observed in many cases of excited action of the heart, especially when there is a defective tension of the arteries, as after great losses of blood, and otherwise where there is a lax state of the vascular system.*

The general symptoms of disease of the aortic valves vary much according to its degree. When slight, it may occasion only more or less palpitation and shortness of breath on exertion, perhaps with a feeling of tightness or pain at the sternum, and other of the common symptoms of moderate disease of the heart. But, when extensive, it is the most formidable kind of valvular disease, and generally proves fatal in the shortest time. I have known several cases run their course in a few weeks after the first complaint had been made of symptoms referred to the heart. It is probable that, in some of these, latent disease had been going on for some time in the valves, and was suddenly aggravated by mechanical violence or inflammation occurring, and to which alone the disease was ascribed. There then come on habitually increased action of the heart and dyspnœa, sometimes aggravated by fits of palpitation and orthopnœa, sometimes with angina, cough, and expectoration, often containing blood. There is commonly some anasarca, more rarely ascites and hydrothorax. Hæmoptysis and dropsical effusions have been too exclusively attributed to disease of the mitral valve and right side of the heart: I have seen them quite as frequently with severe aortic disease; in fact, where the left ventricle cannot effectually unload itself, whether from obstruction or regurgitation, there is just as much interruption to the circulation, pulmonary and venous, as if the mitral orifice or the right heart were diseased; and there is often a greater failure of the capillary circulation, from the impaired tension of the arteries. Among the effects of this, I may mention that regurgitant lesions of the aortic valves are often attended by a pallidity and emaciation, or in more acute cases by pasty puffiness of the integuments, with less colour than is commonly seen in mitral disease; in which the symptoms are more simply those of venous congestion. There is often, too, a restless irritability about the subjects of aortic disease, comporting well with the jerking character of the pulse; whereas with mitral disease it is not uncommon to see an unusual hebetude and torpor. But these distinctions are most uncertain.

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* Dr. Henderson has pointed out another peculiarity in the pulse with aortic regurgitation, that the radial pulse follows that of the heart by an interval longer than usual.

Of the lesions of the *left auricular*, or *mitral valve*, we must now speak. *Obstructive disease of the mitral orifice* consists in its contraction, in the adhesion together of the laminæ of the valve and of their cords, as in fig. 1, or in bodies projecting into it. When this is capable of producing a murmur, it is at the time of the second sound, when the blood passes from the auricle to the ventricle at its diastole; but I believe that a murmur thus caused is of very rare occurrence; perhaps only in case of considerable contraction of the orifice with thickening of the walls of the ventricle, which gives them diastolic elasticity, and thus sucks the blood from the auricle with some force: such was the state of the parts in one out of the only two or three cases which I ever met with, in which the murmur with the second sound was not distinctly referable to regurgitant disease of the aortic valves. Where such a murmur does exist, it would probably be best heard in the same situation as the familiar one of regurgitant disease of the mitral orifice; but its best distinction would be, that it does not modify or supersede the second sound in the arteries, as regurgitant disease of the aortic valves does.

Regurgitant lesions of the mitral valve are attended by a murmur with the impulse and first sound, produced by a sonorous jet of blood through the imperfectly closed valve at each systole.* I have more than once adverted to the nicely-adjusted mechanism of this valve, and I have already given a sufficient number of examples of the lesions by which this mechanism may be deranged. They are very common, and constitute a large proportion of the instances of cardiac murmurs which especially present themselves in women and young persons below the age of twenty. Now where may this murmur be best heard? The mitral valve is situated about the cartilage of the fourth left rib near the sternum; but the spongy lung, and the right ventricle too, if it be dilated, intervenes between it and the walls of the chest; hence the murmur is seldom transmitted opposite to the valve so well as through the apex, to which the fleshy columns of the valves are attached, and which, at the time that the murmur is produced, is in close contact with the walls of the chest, somewhere between the fourth and seventh ribs below, or a little in front of the left mammilla. Here accordingly I have generally found mitral murmurs most distinct, being often quite superficial, and so loud as to eclipse the natural double sound; and this being still audible at the upper and lower ends and to the right of the sternum, and in the carotid arteries, where the murmur is inaudible or distant, completes the diagnosis. Great enlargement of the heart, or consolidation of the lung, sometimes transmits the murmur loud to walls higher up, in which case the dulness on percussion at the spot would explain the unusual circumstance. In a few instances I have heard a mitral murmur very loud and superficial about the third rib, above the mammilla, and I have been led to suspect that it is in these cases transmitted by the dilated auricle which receives the refluent jet of blood. It is sometimes also very audible in the left back, and below the axilla. The character of mitral murmurs is generally blowing, but sometimes passing into a whistle, and more rarely grating. In some cases it accompanies the whole systolic act, and terminates with the second sound: in others it is confined to the end of the first sound, giving to it an additional vowel or a roughness, as we may express by the words *lōō-dŭp* or *lurr-dŭp;* or, if confined to the beginning, *rrhub-dŭp* or *jub-dŭp*. These murmurs are not always constant, especially when the action of the heart is irregular or unequal. We then often hear some of the beats with the murmur, and others without it. Sometimes the murmur alternates with a double first sound, which I have compared to the footsteps of a cantering horse, and which I have conjectured to depend, in such instances, on a loose state of part of the mitral valve.

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* Dr. Elliotson was the first who published the fact that regurgitation through the mitral valve causes a murmur; but he states that he heard of it from Dr. J. Johnson. Dr. Hope claims it as a discovery of his own, made in 1825.

As there is, in most cases of considerable disease of the mitral valve, more or less hypertrophy and dilatation of the heart, the signs of these conditions may be added to those of the valvular lesion. But if the regurgitation is free, it may much diminish the loud sound of dilatation, and the strong impulse of hypertrophy of the left ventricle, by removing the resistance offered by the closure of the mitral valve. Hence the tightening flap of the ventricular sound may be partially lost backwards into the auricle, and followed by a heavy diastolic collapse, when the blood flows in again from the distended auricle. When there is contraction of the mitral orifice, there is usually more hypertrophy than dilatation in the left ventricle, with a strong impulse over it, and the blowing of regurgitation, which is scarcely ever absent, even in these cases. The other compartments are dilated, and may give, as usual, the short double sound.

The pulse varies very much in mitral disease. In many cases, particularly those in which there is constriction of the orifice, it is very irregular and unequal. It is generally weak also, particularly where there is either much constriction or free regurgitation. Yet I have known cases in which the murmur at the apex, during life, and the appearances after death, showed that the regurgitation must have been free, and the pulse was nevertheless strong and jerking, there being also dilated hypertrophy. And when the heart acts irregularly, the strong beats of the pulse are in some instances attended with a louder murmur at the heart than usual. In other instances again, where the pulse was intermittent, I have observed each intermission or weaker beat to be accompanied by a loud blowing at the apex, which was much less distinct in the other beats. This intermission was obviously from the force lost backwards in regurgitation, the cause of which was probably moveable, or subject to variation, as in the case of a partial looseness causing retroversion, or of an excrescence between the laminæ.

The general symptoms of mitral disease, both obstructive and regurgitant, are, besides those of inordinate and irregular action, those especially of pulmonary and venous congestion. Sometimes the disease appears to be more pulmonary than cardiac, the patient being asthmatic in paroxysms, or suffering habitually from dyspnœa, with cough, and copious thin mucous expectoration sometimes containing blood. In other cases, especially those of a slighter kind, the lungs suffer little, and the chief symptoms arise from a congested state of the venous system; such are enlargement, pain, and deranged action of the liver, hæmorrhoids, scanty and disordered urine, ascites, hydrocele, and anasarca, congestive headaches, giddiness, &c. These occur in their greatest degree when the right ventricle is considerably diseased; but in a slight extent I have often met with them when the patient made little or no complaint of the heart, and when the chief sign of its disease was a constant murmur at the apex, indicating regurgitation through the mitral valve. When this defect does not obviously derange the action of the organ, it may still affect other organs through the circulation: continually putting back, at every beat, a little blood into the veins, may produce an accumulative effect in the different organs of the body, and none are so likely to suffer as those largely supplied with venous blood, and have been previously liable to any derangement. There are few cases of diseased heart, especially when this valve is affected, in which the functions of the abdominal viscera are not occasionally disordered; and I repeat, that this disorder is in many cases more prominent than the organic defect from which it has arisen. In the worst forms of mitral disease, the conditions of pulmonary congestion and venous obstruction are combined; and then we see the terrible array of symptoms, which has rendered the name of organic disease of the heart so appalling, yet which does not occur in above one in a score of the cases where the structure of the organ is really diseased.

Of the signs of disease of *pulmonary semilunar valves* I know nothing from *post mortem* experience. They are so rare, that I have only met with three cases in which I have suspected its existence during life. These presented a slight blowing or grating murmur with the first sound, audible at the middle of the sternum at its left margin opposite the valve, and below, over the right ventricle, but not below the left breast, at the top of the sternum or in the carotids; and as the pulmonary artery immediately plunges backwards under the arch of the aorta and into the lungs, it presents no course over which the sound could be traced. Speaking only *à priori*, of *regurgitant* lesions of this valve, if they could be distinguished at all, it would be by a murmur with the second sound heard at the middle of the sternum, and not in the arteries, where the natural second sound would be unimpaired, and there would be none of the peculiarities of pulse which attend such a murmur when connected with diseased aortic valves.

Of the *right auricular* or *tricuspid valve*, I can speak only as to its *regurgitant* lesions. We have before found that regurgitation through this valve is probably very common; it is chiefly known by pulsating swelling of the jugular veins, and is not commonly attended with any audible murmur. This, I think, may be explained by the laminæ of the valve being so yielding as not to resist the current firmly enough to produce sound, or if a slight sound be produced it is lost backwards. But in a few cases I have heard a deep blowing or grating murmur, distinct from the middle to the bottom of the sternum, in the epigastrium, and at the margin of the ribs close to it, not audible under the left breast, at the top of the sternum, nor in the carotid arteries, and accompanied by pulsating swelling of the jugulars, and signs of dilated hypertrophy of the right ventricle. This murmur I have been induced to ascribe to regurgitation through a tricuspid valve, thicker and more rigid than usual; and in two such cases I have found this condition of the valve after death: in one of them with some constriction of the orifice. Both these cases were combined with great dilatation and hypertrophy, especially affecting the right ventricle and auricle, and the symptoms had been the ordinary ones of extensive organic disease of the heart, affecting the lungs with unusual severity.

The different lesions of the valves and orifices which I have been describing may be variously combined; in which case their diagnosis will be more difficult, for the positive characters of each are seldom so distinctive as to announce them clearly. Murmurs may be produced in two orifices at once—as by obstructive disease of the aortic and regurgitative disease of the mitral. The former might be recognised by the grating sound in the arteries, but the mitral murmur could not be distinguished from it, unless it were very different in character; as when it is whistling or whiffing, and quite superficial at the apex. On the ground of such differences I have several times founded diagnoses of lesions of both orifices, but I do not lay much stress on them. The physical diagnosis of valvular disease is in some instances very easy, but it is far from being so always; and in the more complicated cases, accompanied by irregular action, without any definite or well-localized murmur, it sometimes must be too conjectural to be depended on.

Various structural Diseases of the Heart.

Softening of the heart, as of other organs, may arise either from inflammation, from defective nutrition, as from obstruction of the coronary arteries, and probably also from a diseased state of the blood. To the latter cause, perhaps, may be ascribed the softening described by Dr. Stokes and others, as affecting the heart in the advanced stage of adynamic fevers.

From whatever cause softening may proceed, it undoubtedly impairs the strength of the heart's action, and therefore the strength of the impulse, and, as Laennec supposed, the loudness of the sounds. Dr. Stokes has observed this to be so much the case in the fevers alluded to, that the first sound is sometimes inaudible. I have found it much impaired, but never inaudible. In a few cases I have suspected softening in persons who complained much of short breath, oppression, and faintness, on exertion, with a frequent, feeble, and sometimes irregular pulse, and in whom there was not the loud sound of dilatation, the strong impulse of hypertrophy, or the extended dulness of a heart loaded with fat. The softening in such cases seems to be unconnected with inflammation. In one instance I have known it accompanied by anginal paroxysms, and prove suddenly fatal by rupture.

Symptoms and signs not unlike those of the last-named lesion, are sometimes produced by *fatty accumulations* around the heart, particularly in females; but the increased dulness under and to the left of the lower and mid sternum often shows, in this case, that there is an addition to the natural size of the organ, the defective impulse and strength of pulse distinguishing this from muscular hypertrophy.

True *sebaceous degeneration* of the heart differs from the preceding affection, in the fatty matter being deposited in, and not on the muscle of the organ, which thereby becomes changed in nature and impaired in function. The tissue is paler than usual and easily torn, and on being pressed between paper gives it a greasy stain. The origin of this affection is quite obscure. It has been met with in cases of dropsy and cerebral apoplexy.

Ossification of the heart is less rare than the last affection, but it seldom amounts to more than osseous patches in the mitral or aortic orifices. I have met with a case where the base of the left ventricle was encircled by an osseous zone, from half to an inch and a half broad, extending also around the mitral orifice. The coronary arteries were also ossified, and the substance of the heart enlarged and softened. A very similar case has been described by Mr. Smith of Dublin. These cases presented the common symptoms of organic disease of the heart; in my instance, with paroxysms of angina. Instances of more extensive ossification have been described by Corvisart, Burns, Renauldin, &c.

Cartilaginous and fibro-cartilaginous deposits are now and then met with under the pericardium, and in a few rare examples have been found invading the muscular structure. Some cases of this description which I have seen have exhibited other marks of previous inflammation, such as adhesion of the pericardium, opaque thickening of the endocardium or valves. I have reason to think that they, as well as the much commoner fibro-cellular patches, originate in inflammation, or in a process akin to it.

Rupture of the heart was supposed by Laennec to be a consequence of ulceration of the interior; but Dr. Townsend has shown that the most common situation of the rupture, the left ventricle near the apex, is rarely the seat of ulceration. It is more probably the result of partial softening with hypertrophy of other parts. Death has in most instances been immediate, with anginal syncope.

Malignant disease rarely affects the heart: a few instances are recorded by French authors. I have seen the septum to a great extent converted into a medullary tough substance with a nodulated surface, which seemed to me of a carcinomatous character. The patient had died suddenly.

Polypous or *fibrinous concretions* in the heart were long supposed to be common causes of serious and fatal impediment to the circulation; and although morbid anatomy has shown that in most instances they are formed either only after death, or during the failure of the circulation for a short time before, yet it is probable that sometimes their formation is, as Laennec supposed, the immediate cause of death in diseases of the organs of circulation and respiration. Of the coagula found in the heart and great vessels after death, those only can be supposed to be *true*, or formed before death, which are wholly fibrinous, firm in texture, yellowish white or pinkish, and partially adherent to the lining membrane, which itself may be more injected than usual. I may state as another proof that a concretion has been formed before death, when the valvular tendons pass through it by smooth apertures, much larger than themselves, showing that they had moved during its formation. Laennec says that we may diagnosticate such a polypus, when the motions and sounds of the heart, before distinct and regular, become so irregular, obscure, and confused, that they cannot be distinguished. Bouillaud adds the signs of tumultuous pulsations, and a blowing, hissing, or musical murmur, with orthopnœa, lividity, extreme anxiety, cerebral congestion, and other symptoms of suddenly obstructed circulation. Such symptoms and signs have been rarely known to come on and pass off again. I met with them the day before death in a case of chronic pericarditis, with regurgitant mitral disease; but I found no polypus in the heart; only a thin loose coagulum. From the numerous researches of the late Dr. Mackintosh, it is probable that the symptoms of collapse in malignant cholera are partly due to the formation of large long fibrinous concretions in the heart and great vessels.

Malformations of the Heart.

The most common as well as the most important malformations, in a pathological point of view, are those which occasion the distribution of venous with, or in place of, arterial. Some of these are evidently the result of erroneous conformation, and which is as little intelligible as normal conformation: such is transposition of the aorta and pulmonary artery, the latter arising from the left, the former from the right ventricle. An opening in the septum of the ventricles may equally result from an arrest of the growth of this part, or from a morbid obstruction in one of the arterial orifices, which may be the consequence of disease during fœtal life. A similar morbid cause may lead to the permanent patency of the foramen ovale and ductus arteriosus, which is of all malformations the most common. Such morbid causes may sometimes operate before birth; but a case has occurred under my observation that has convinced me that malformations of the last-named kind may originate after birth, from circumstances which interfere with the due performance of respiration, and which therefore interrupt the circulation through the lungs.

Malformations even of great extent are not incompatible with life; but they lower the activity of the functions towards the standard of reptiles and other animals, whose normal state of circulation they resemble. It has been much discussed of late years, whether cyanosis, or the blue discoloration of the skin often met with in connexion with malformation of the heart, be dependent on the mixture of venous with arterial blood, or on the obstructed circulation arising out of the same cause. Certainly malformations have existed without cyanosis; but as far as my experience or reading goes, cyanosis, in its full extent, never occurs without malformation, and I think that the circulation of black blood in the arteries is its essential cause, that cause being exaggerated by obstructions to the circulation, which not only increase the flow of venous blood into the arteries, but make more manifest its dark colour on the surface of the body by distending the capillaries. For further views and facts on this subject, I must refer to the article *Malformations of the Heart*, in the Cyclopædia of Practical Medicine, and to the able summary of Dr. Joy, in the Library of Practical Medicine.

I am not aware of any distinctive physical signs of malformations of the heart. Those causing partial obstructions at the arterial orifices would cause the usual systolic murmurs distinguishable, as explained under the head of valvular disease. A deeper and hoarser murmur might be produced by interventricular openings, provided the strength of one ventricle be such as to cause a forcible current into the other, and such a murmur would probably be best heard about the mid-sternum, and not in the arteries of the neck. Possibly an open foramen ovale might be the seat of an obscure murmur of a similar kind.

CHAPTER V.

DISEASES OF THE GREAT VESSELS.

Functional and inflammatory diseases of the great arteries.

VERY little is known concerning the temporary derangements of the aorta and pulmonary artery, and this is scarcely to be wondered at, when we consider that the physiological properties of these vessels are little known to the semeiologist. I have ascertained that direct irritation, by galvanism or by a mechanical stimulus, causes the contraction of arteries; but it is yet unknown how far the same effect may be produced by sympathetic irritation in the living body, and whether any opposite state can produce an unusual relaxation of these vessels. The phenomena of arterial pulsation, sometimes so remarkable in the carotids and abdominal aorta, appear to depend on a defective tone of these vessels, and have been by Laennec and others considered to arise from modified innervation; by others, to be the result of undue distension; and by a third party, among whom Dr. Stokes may be named, as sometimes connected with inflammation of parts beyond. This last view receives support from the well-ascertained fact, that arteries leading to an inflamed part become enlarged, and pulsate with unusual force. But other causes, short of inflammation, suffice to cause increased pulsation, and they all probably do so by either obstructing the branches of the artery beyond, or by impairing the tonicity of its coats.

Morbid pulsation of arteries is attended with a great increase of sound as well as of motion. The sound in the carotids and other arteries near the heart is commonly double, and unaccompanied by a murmur, unless there be some valvular disease, or partial constriction or pressure on the artery. A very slight pressure is sufficient to cause a murmur, and in the case of abdominal pulsation it is very difficult to avoid so pressing with the stethoscope. The most useful distinction between arterial pulsation and aneurism is, that in the former the increased motion and sound are not confined to one spot, but extend along the vessel, and reach to the neighbouring branches.

Aortitis is scarcely known but by its anatomical characters. Some morbid anatomists have confounded with inflammation the red stain often exhibited by the interior of the arteries and heart: this is simply an imbibition of tissue with colouring matter, and may be distinguished from the redness of inflammation by not being vascular. The vascularity of inflammation is always more or less obvious on close inspection, particularly with a lens. Inflammation of the aorta is commonly in patches, and may lead to the deposition of lymph, and to softening. According to Bizot, these changes lay the foundation of the opaque tough patches often seen in the aorta. These patches sometimes are within, and sometimes without the lining membrane. Bizot distinguishes betwixt them and the softer, less organized spots, which are commonly called atheromatous, and states that the latter, and not the former, become ossified. It appears to me that the views advanced in the preceding pages with regard to the products of pleurisy, and the formation of tuberculous matter, are applicable to this subject; and that the atheromatous and calcareous deposits of arteries are the products of degraded nutrition, resulting in some cases from low forms of local inflammation, in others from constitutional defect. In aged persons we find these changes in the arteries prevailing to a great extent; and like ossification of cartilages, and wasting of the more vital tissues, they are to be viewed as the marks of natural decay, by which organized beings are limited to a space of years. There can be little doubt that some of these modifications of nutrition lay the foundation of other alterations which may more speedily destroy life by altering the mechanism of the circulation. These we shall now briefly consider.

Dilatation and aneurism of the aorta.

The aorta, from its origin to its descending portion, is liable to become dilated, and the dilatation or aneurism may consist in an equal distension of all its coats, which is *true* aneurism, or in a distension of the outer coat, the inner and middle coats being ruptured, which is *false* aneurism. The latter is, however, the commonest kind recognized as aneurism, true aneurism seldom being sacculated, but causing a more general dilatation of a portion of the whole tube. It will be more convenient for diagnostic purposes to distinguish the two kinds, *general dilatation* and *aneurism.*

General dilatation of the ascending aorta and of its arch frequently accompanies disease of the left ventricle, especially that which affects the aortic valves. It may occur with very little apparent disease in the coats of the artery, and is then more uniform; but more commonly these are much altered, and then the dilatation is more irregular, and with a tendency to form lateral sacs, particularly near the attachment of the aortic valves and below the branching off of the innominata. The alterations of the coats consist in various deposits of opaque matter, differing in consistence and appearance, sometimes softer than the rest of the tissue, and easily separated from the serous coat; in other instances harder and tougher, involving the middle fibrous coat: in either case, there being occasionally osseous scales or concretions in them, and sometimes the whole caliber of the vessels being beset with all these deposits, broken up into the most rugged irregularities.

The aorta thus dilated, and wanting its proper regulating elasticity, receives the contents of the ventricle with more abruptness than usual; this circumstance, with the greater size of the vessel, increases the force and loudness of the pulsations at the upper part of the sternum, above it, and in the carotids. The character and sound of the pulsation will depend on the state of the walls of the artery and of its orifice. If the orifice be not contracted, nor the valves indurated, nor the ventricle much enlarged, nor the walls of the artery very rough or with projecting points, the pulsations will be simply unusually strong, with a double sound; the first sound being exaggerated by the impulse of the blood against the dilated walls of the artery, the second being that of the aortic valves simply transmitted. But if any of these conditions be present, they will constitute a resistance to the moving current of blood, and a murmur, more or less harsh and grating, will be the result, often with a thrill perceptible to the touch. To distinguish whether this murmur is produced in the orifice or in the rugged artery, we must return to the heart, and seek for the signs of obstructive disease of the aortic orifice. If they be not found distinct at the mid-sternum, whilst the first sound in the upper sternal region, and over the carotid arteries is deep, loud, and harsh, the probability is that it is produced in the artery; and even if there be signs of diseased aortic valves—when the murmur in the arteries is single, and much louder and more harsh than that heard at the middle of the sternum, and attended with increased pulsation—

there is still probably disease of the aorta, which transmits its own sound rather than those of the heart. Dilatation of the aorta above the orifice also increases the murmurs resulting from partial impediments in the orifice; and when the ventricle is also enlarged, the natural orifice becoming *relatively* narrow, may be the seat of a murmur without any real constriction.

Aneurism, *partial* or *saccular dilatation* of the ascending or bending portion of the aorta, or of the innominata or the other great arteries, may cause additional signs and symptoms, in consequence of the greater local extent of their tumor. When they are yet small, they may only increase the impulse and sound of the top of the sternum, and perhaps be accompanied with some general symptoms of disordered circulation, and, according to Dr. Billing, a peculiar resilience of the pulse; but as they increase in size, they press on the adjoining parts, and cause more or less disorder of their functions, and produce many physical signs. Thus their pulsating pressure on the pneumogastric nerves may sometimes, at an early stage, disorder the sensations of the lungs and stomach, causing feelings of slight dyspnœa, cough, nausea, or gastrodynia: increasing bulk of the tumours may compress—the veins—hence their distension, with lividity, congestion and œdema of the parts from which they come, the head, neck, and upper extremities; the nerves—hence pain, numbness, and paralysis of the arms, hands, and back of the neck; spasm of the glottis and alteration of the voice; the œsophagus—hence difficulty of swallowing; the trachea or its branches—hence bronchitis, severe cough often in paroxysms and dyspnœa; the bones and cartilages—hence caries and absorption; and in case of the spine, perhaps disturbance of its contents; and lastly, some of the other arteries—whence deficient pulse and circulation in the part to which the obstructed artery leads, occasionally one of the upper extremities. Some or other of these symptoms are generally present in the progress of aortic aneurisms; and their number and intensity may be generally explained by the particular position and size of the tumor.

The physical signs of aneurism of the upper part of the aorta are those of increased pulsation and sound, and, in advanced stages, those of a pulsating tumor presenting itself in various positions. The early increased pulsation may not differ from that of a generally dilated aorta, except, perhaps, that it is perceptible more at one spot above the clavicle, or that its sound is more to one side than towards the middle of the sternum. The sound may be like that of general dilatation, either simply double, like that of the heart, or single, attended with a grating or whizzing; the second sound of the heart being inaudible or obscure. The same remark will apply to more advanced aneurisms. They are sometimes accompanied by a grating single or double sound at each pulse, but often there is no grating; both the sounds are loud, and give one the idea that the heart is beating under the top of the sternum. In fact, that aneurisms shall produce a grating, there must be more than simply a sac smoothly communicating with the artery; the edges of the orifice must be abrupt and projecting, or contracted; or there must be some roughness or prominence in the artery near it; or there must be some other vent out of the sac, so that the current may not beat *in* merely, but *whiz through.* If the sac be very elastic, and the orifice small, there may possibly be a jet of blood to-and-fro, in and out of it, at each pulse and interval, causing a double whizzing; but I have never seen such a case. In most of the cases which I have seen, there has been at the top of the sternum a short grating sound, or a loud double sound, alternated with a duller, more prolonged sound, which I suppose to be seated in the compressed veins. To distinguish such sounds from those of diseased aortic valves, we must have recourse to the same expedients as for the diagnosis of general dilatation; only, in case of aneurism, the sounds may be more local, and not so strongly transmitted to both carotids in the neck. When the aneurism reaches such a size as to form a tumor, perceptible above the sternum or clavicle, or between the upper ribs close to the sternum, the only remaining difficulty is to distinguish it from other tumors: other tumors may produce the same symptoms and signs of pressure, and from the beating of the artery under them they may seem to be pulsating; nay, by compressing it or one of its branches, they may cause a grating or whizzing at each pulse, very like that of aneurism; and from the same cause they may disturb the action of the heart. But the pulsation of an aneurism may be generally distinguished by its greater extent, its distension in all directions, and sometimes by its softness; its sounds are generally much louder; and the pulsations of the arteries beyond, although occasionally unequal, are generally more free than they would be, if a solid tumor were pressing much on their exterior.

Aneurisms of the descending aorta may generally be known by an increased sound of pulsation, which may be grating or whizzing, at some point along the left of the dorsal spine. As it advances and forms a tumor forwards, these phenomena will be obvious in front; and it may produce many other signs and symptoms, by pressing and displacing the lungs and the heart. If it increase backwards, it causes absorption of the ribs and vertebræ, producing paralysis and other symptoms of pressure on the spinal marrow, and presenting its pulsating tumor behind.

Aortic aneurisms may prove fatal by suffocation, by causing inanition, by pressure on the air-tubes, and by pressure on the œsophagus or thoracic duct, or by hæmorrhage from the ultimate rupture of the sac. In rare instances they have terminated in spontaneous cure, by becoming filled with fibrinous coagula, which subsequently contract and become organised. The directions in which aneurisms may open are very numerous, and various results may ensue; into the lungs or air-tubes; into the cavity of the chest; into the œsophagus; outwardly; into the pericardium; into the spinal canal; into the vena cava; into the pulmonary artery, right ventricle, or one of the auricles. In the last four cases the rupture may not be immediately fatal; and there may result a complicated disorder of the mechanism of the circulation, more or less anomalous, and baffling the application of diagnostic rules. In a case where the opening was into the right ventricle by two small orifices, a loud grating murmur was heard with the first sound between the cartilages of the second and fourth left ribs; and I was led to attribute this murmur to an aortic aneurism, and not to valvular disease, because, although systolic and single, it was prolonged beyond the occurrence of the second sound of the heart, and consequently continued during the diastole of the ventricles.

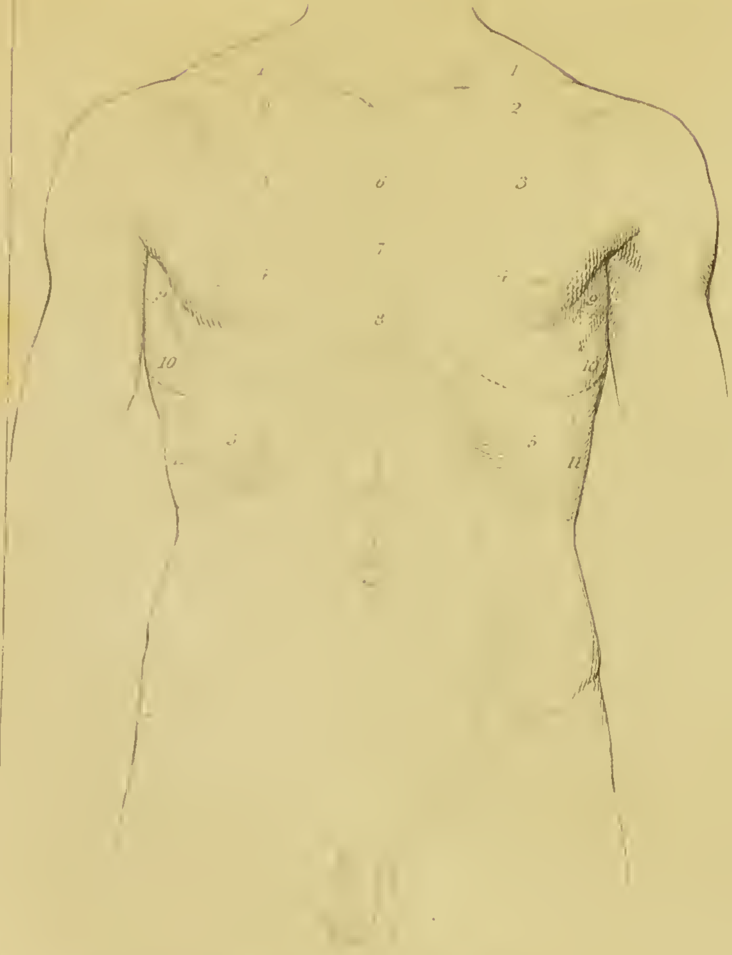
Dilatation and aneurism of the pulmonary artery is extremely rare, and nothing has been ascertained with regard to its signs. They would probably be those of obstructive disease of the pulmonary orifice, together with increased pulsation to the left of the sternum in the second and third intercostal spaces.

TABLE I.

Tabular View of the Thoracic Regions, in relation to the signs of Auscultation, &c.

SEE PLATE II.

REGIONS. PLATE II.	NUMBER.	SITUATION.	NATURAL STROKE SOUND.	INTERIOR CORRESPONDING PARTS.	SIGNS MOST COMMONLY PRODUCED THERE BY DISEASE.
1. Supra-clavian or top front.	Right & Left.	Above the clavicle.	Pretty clear in thin persons by mediate percussion.	Summit of the lungs.	Dulness in phthisis. Clearness and prominence in emphysema. Venous and carotid murmurs.
2. Clavicular.	Do.	Clavicle.	Very clear and pulmonary towards the sternum: clear to the middle; dull close to the humerus.	Front top of the lungs.	Dulness on percussion in phthisis; generally most on one side.
3. Infra-clavian Upper front.	Do.	Between the clavicle and lower edge of third rib.	Very clear.	Superior lobes of the lungs; large bronchi near the sternum.	Irregular dulness, diffuse bronchophony, impaired or altered breath sound, and afterwards cavernous rhonchus and pectoriloquy, in phthisis. Various rhonchi in catarrhs.
4. Mammary Middle front.	Do.	Between the upper edge of the fourth and seventh rib.	Very clear; particularly by mediate percussion. Less clear below edge of pectoral muscle on right, and near sternum on left side.	Middle lobes of the lungs; large bronchi in the upper part. The heart almost covered by the lungs, between the left breast and lower sternum. The liver also covered in right lower part.	Rhonchi in catarrhs; more rarely phthisical symptoms. On the left side, dulness in pericarditis and enlargement of the heart; increased impulse in hypertrophy, and increased sound of pulsation in dilatation of the heart; murmurs in valvular disease.
5. Infra-mammary. Lower front.	Do.	Between the seventh rib and the margin of the cartilages of the false ribs.	Dull on the right side: on the left irregularly dull, or unnaturally resonant.	The liver on the right, and the stomach on the left side, covered only on the upper part by the thin margin of the anterior inferior lobes of the lungs.	Crepitant rhonchus in incipient pneumonia. Extinction of respiration in advancing pleurisy. Dry crepitation in interlobular emphysema. Friction sound in dry pleurisy.
6. Upper sternal.	1	Upper third of the sternum.	Very clear.	Trachea and large bronchi, and blood vessels.	Bronchial rhonchi. Only half the sternum dull on percussion in hepatisation; the whole dull in extensive liquid effusion of one side. Signs of diseases of the right side of the heart; dulness on percussion in effusion or fat, in the pericardium, enlarged heart, &c.
7. Mid. sternal.	1	Middle third of the sternum.	Clear.	Margins of the middle lobes of the lungs. Base of heart and great vessels.	
8. Lower sternal.	1	Lower third of the sternum and ensiform cartilage.	In the upper part clear; less so in fat persons. Below, sometimes more dull; sometimes tympanitic.	Above margins of the lungs; below the heart and liver.	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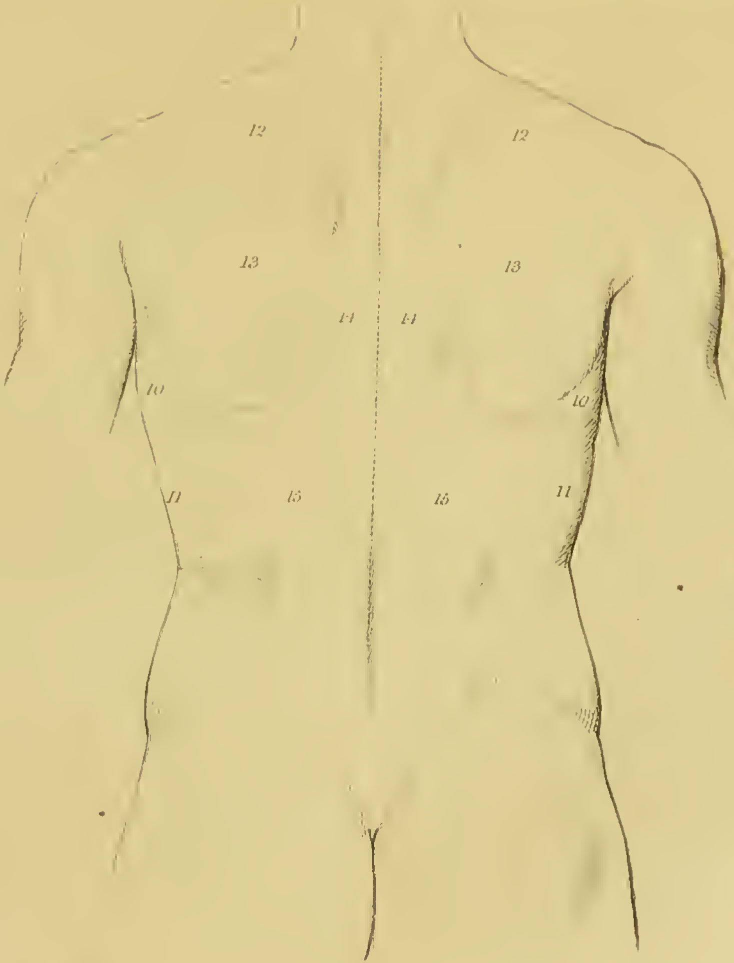
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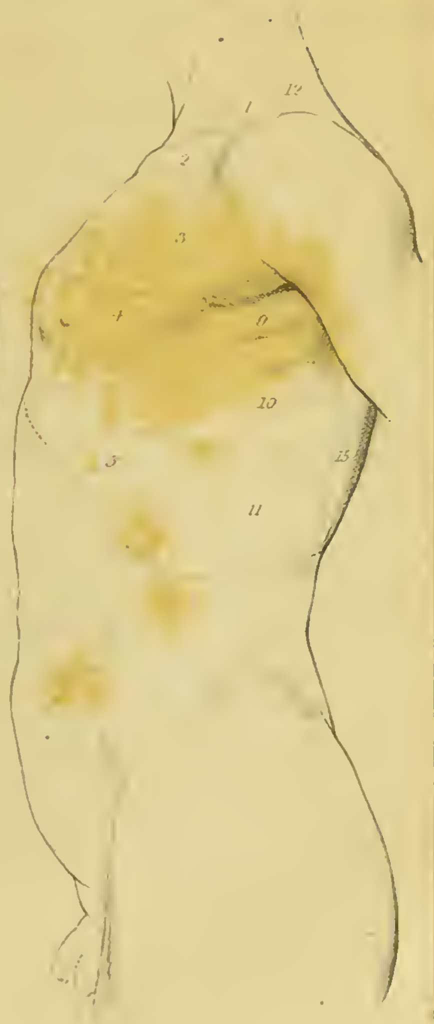
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Regions. Plate II.	Number.	Situation.	Natural Sound on Percussion.	Interior Corresponding Parts.	Signs most commonly produced there by disease.
9. Axillary. Upper side.	Right & Left.	In the axilla above the 4th rib.	Very clear.	Upper side of summit of the lungs. Large bronchi.	Dulness on percussion, cavernous rhonchus, pectoriloquy, &c. in phthisis. Catarrhal rhonchi.
10. Lateral. Mid. side.	Do.	Between the fourth and seventh rib at the side.	Very clear; unnaturally so, in emphysema of the lung.	Middle of the lateral lobes of the lungs.	Dulness on percussion in advanced pleurisy; and on the right side, from enlarged liver. Ægophony in advancing pleurisy; crepitant rhonchus, and bronchophony in advancing pneumonia.
11. Inferior lateral. Lower side.	Do.	Below the seventh rib at the side.	The same as the infra mammary.	Margin of the lateral lobes of the lungs; the liver on the right side; the stomach and spleen on the left.	Crepitant rhonchus in incipient pneumonia. Extinction of respiration in pleurisy.
12. Suprascapular. Upper back.	Do.	Above the spine of the scapula, and third dorsal vertebra.	Dull by direct percussion. A tolerably clear sound may be elicited by mediate percussion, particularly on the spine of the scapula.	Posterior part of summit of lung.	Dulness on percussion in extensive tubercular accumulation; cavernous rhonchus and respiration, and pectoriloquy in phthisis. Catarrhal rhonchi.
13. Scapular.	Do.	The scapula below its spine.	The pectoral resonance can be elicited from this region only by mediate percussion.	Middle posterior lobes of the lungs.	Bronchial sounds. Ægophony in pleurisy. Bronchophony in pneumonia.
14. Interscapular. Mid. back.	Do.	Between the inner margin of the scapula and the spines of the vertebræ.	Pretty clear by mediate percussion, or when the arms are crossed, and the head bowed forwards. The spinous processes of the vertebræ sound well.	The roots, and inner parts of the posterior lobes of the lungs.	Bronchial sounds. In the upper part, breath and voice sounds never destroyed in effusions into the pleura. In the lower portion sometimes ægophony in pleurisy, crepitation and bronchophony in advancing pneumonia. Signs of diseased bronchial glands.
15 Inferior dorsal. Lower back.	Do.	Below the inferior angle of the scapulæ, and seventh dorsal vertebra to the level of the twelfth vertebra.	Clear in the upper portion, by striking on the angles of the ribs, or by mediate percussion. Below, duller on the right, and tympanitic on the left side.	Base of the lungs. The liver encroaches on the right, and the stomach and intestines on the left side.	Crepitant rhonchus and bronchophony in incipient pneumonia and œdema; ægophony in pleurisy, and dulness on percussion in both.

TABLE II.

TABULAR VIEW

OF THE

MOST CHARACTERISTIC SIGNS OF THE PRINCIPAL DISEASES OF THE LUNGS AND PLEURA.

Disease.	Physical Signs in the Part Affected.			Sputa.
	Stroke Sound	Breath Sounds.	Voice Sound.	
Acute Bronchitis.	Sometimes slightly diminished.	Weak in parts; accompanied by a rhonchus, first sonorous or sibilant, afterwards mucous. Towards the end sometimes inaudible in parts.	Natural.	Mucous; at first thin, then viscid; at last opaque.
Chronic Bronchitis.	Slightly impaired, if the disease is extensive.	Weak in parts, or irregular, with mucous rhonchus.	Natural.	Opaque mucus, sometimes purulent, sometimes streaked with blood.
Dilated Bronchi.	Partially impaired, sometimes tubular.	Bronchial, or a gurgling rhonchus.	Bronchophony or even pectoriloquy.	The same: sometimes fœtid.
Bronchorrhœa.	Partially impaired.	Weak with sonorous, sibilant, and mucous rhonchi.	Diminished.	Watery.
Bronchial Congestion.	Natural.	More or less extinct in parts. Occasional, sibilant, sonorous, and dry mucous rhonchi.	Diminished.	A scanty pearly thick mucus.
Spasmodic Asthma.	Sometimes impaired.	Weak, or even inaudible; but distinct and even puerile, immediately after holding the breath a while.	Diminished.	Sometimes thin & copious at the end of each paroxysm
Peripneumony 1st stage.	A little impaired.	Weak with crepitant rhonchus.	Natural.	Viscid, of a rusty hue.
2d stage.	Quite dull: tubular near root of lung.	Bronchial, with crepitant rhonchus.	Bronchophony	Rusty & very viscid, or none.
3d stage.	The same.	Bronchial, sometimes a coarse mucous rhonchus.	None.	Sometimes purulent or brown and watery.
Emphysema.				
Flaccid.	Too clear.	Loud; sometimes whiffing.	Natural.	
Rigid.	Clear: tone raised.	Diminished, and sometimes almost extinct. Cough accompanied by sibilant rhonchus.	Diminished.	A thick dirty grey mucus.
Œdema.	Dull, if the effusion is extensive.	Weak, with subcrepitant rhonchus.	Sometimes slight bronchophony.	Watery and mucous.
Pulmonary Apoplexy.	Dull, if near the surface, and extensive.	Extinct in the apoplectic spots; a rhonchus, first subcrepitant, afterwards mucous, around them.	Occasionally bronchophony.	Blood, or bloody mucus.
Pleurisy.	Dull below; often clear or tubular above.	First weak; afterwards extinct, except at the root of the lungs, or adhesions.	At first ægophony; afterwards none, except with adhesions.	None: or catarrhal.

TABLE II. CONTINUED.

DISEASE.	PHYSICAL SIGNS IN THE PARTS AFFECTED.			SPUTA.
	STROKE SOUND	BREATH SOUNDS.	VOICE SOUND.	
Pleuropneumonia.	Quite dull.	At first weak, with crepitant rhonchus; then bronchial, afterwards extinct.	Buzzing ægophony.	As in peripneumony.
Hydrothorax.	Quite dull below; clear above.	Weak or extinct, according to the quantity of the effusion.	Ægophony, when the effusion is scanty.	Various.
Pneumothorax, with perforation of the pleura.	Dull in the dependent parts; very clear above.	Respiration and cough, sometimes amphoric or tinkling. Succussion of the chest produces sound of fluctuation.	Metallic tinkling.	Phthisical.
Phthisis.				
a. Stage of gray induration, and crude tubercles.	Impaired, if the accumulation be extensive.	Weak, with loud expiration; or somewhat bronchial, if the accumulation be extensive.	Diffused bronchophony, if the accumulation be extensive.	Sometimes catarrhal and scanty, sometimes watery, sometimes bloody.
b. Stage of evacuation of the softened tubercles.	Unequal. Generally impaired under a clavicle.	Cavernous rhonchus, respiration, and cough.	Pectoriloquy, when the cavity is empty. Metallic resonance when the cavern is large.	Muco-purulent; purulent; tuberculous curdy matter; sanguinolent, gray or brownish grumous liquid.

TABLE III.

MARKING THE RELATIVE PERIODS AND DURATIONS OF THE NATURAL AND MORBID MOTIONS AND SOUNDS OF THE HEART AND BLOOD-VESSELS.

	Time of a Pulse divided into five equal parts.				
Motions.	Aur. Syst. Ventricu Impul	lar Systole. se.	Diastole.	Re	st.
Sounds Natural.	First	Sound	2d Sound	Sile	nce.
Morbid.	Perica	rditic F	rictio	n	
Murmurs	Obstructiv	e Aortic	Regurgita	nt Aor	tic
	Obstructiv	e Pulmonary	Regurgita	nt Pulmon	ary (rare)
	Regurgita	nt Mitral	Obstr. Mitr	al (rare)	
	Regurgita	nt Tricuspid	Obstr. Tricu	sp. (unknown)	
	Ar	teri	al	Murm	urs.
	Ve	nou	s Mu	rmu	rs.

EXPLANATION OF TABLE III.

The period from the commencement of one pulse to the commencement of another is divided into five equal parts. Of these the two first are occupied by the ventricular systole with the first sound. One-fifth is occupied by the diastole, in part of which occurs the short second sound. The period of natural rest and silence succeeds, occupying the remaining two-fifths. In the columns below are expressed the cotemporaneous morbid sounds, the periods of the pulse at which they must occur, and by the length of the words it is intended to express the duration which they may exhibit in relation to the periods of the whole pulse, and to the natural motions and sounds. Thus, an obstructive aortic murmur may occupy the whole period of the ventricular systole, it may occupy less, but not more; regurgitant aortic the whole period of the diastole, and more or less of that of the rest also, and thus it may precede as well as follow the second sound. Arterial murmurs may occupy nearly all these periods, although they are loudest at the period of the ventricular systole in arteries near the heart, and a little after it in those which are remote. Venous murmurs may be quite continuous.

TABULAR VIEW OF THE MOST CHARACTERISTIC SIGNS OF DISEASES OF THE HEART.

Disease.	First Sound.	Impulse.	Second Sound.	Situation in which the abnormal sound is loudest.	Radial Pulse.	Percussion.
Palpitation.						
Nervous.	Loud and short.	Strong, abrupt, and limited.	Varying.		Frequent, varying.	Natural.
Plethoric.	Loud and prolonged.	Strong.	Loud.		Full and strong; not always frequent.	Dulness sometimes increased.
Syncope.	Short and flapping.	Very weak.	Obscure or null.		Very weak.	Natural.
Pericarditis.						
Before effusion.	Loud and prolonged.	Strong and abrupt.	Loud.		Strong, various in frequency.	Natural.
With effusion of lymph.	Masked by friction sound.	Tumultuous.	Masked by friction.	Whole region of heart.	Generally frequent.	Increased dulness.
With liquid effusion.	First drum-like, afterwards obscure.	None.	Impaired.		Frequent—often irregular.	Extensive dulness.
Endocarditis.	Loud—double—or with murmur.	Strong and abrupt.	Sometimes double or with murmur.	Varying with the seat of the inflammation.	Frequent—often jarring.	Natural.
Hypertrophy.	Dull and prolonged.	Strong and heaving.	Obscure except above midsternum.		Generally strong.	Increased dulness.
Dilatation.	Loud and short.	Slight, but abrupt and diffused.			Weak.	Increased dulness.
Dilated Hypertrophy.	Loud.	Strong, abrupt, and diffused.	Loud, with diastolic shock.		Variable.	Extensive dulness.
Valvular Diseases.						
Aortic, obstructive.	Accompanied by murmur.	Various.	Sometimes impaired.	From midsternum to top, and in carotids.	Various; often strong and jerking.	
——— regurgitant.			Attended or supplanted by murmur.	At midsternum; sometimes above and below.	Jarring; receding, visible.	
Pulmonary, obstructive.	With murmur.			At midsternum: not above nor in the carotids.	Natural.	
——— regurgitant.			Attended with murmur, natural above midsternum.	Below midsternum.	Natural.	
Mitral, obstructive.		Irregular.	Attended with murmur.	Below left breast.	Often irregular.	
——— regurgitant.	Double, or impaired, with murmur.		Distinct, above midsternum—often inaudible below left breast.	Below left breast.		
Tricuspid regurgitant.	Rarely with murmur.			Below midsternum and in epigastrium.	(Pulse in jugulars.)	
Softening.	Obscure.	Weak.			Very weak and frequent.	Natural.
Fatty Enlargement.	Obscure.	Weak and diffused.	Obscure.		Various.	Increased dulness.

APPENDIX.

I. AN INQUIRY RESPECTING THE MOTIONS AND SOUNDS OF THE HEART,

With experiments performed by the Author in Feb. 1835.

In the second edition of this work, published in 1833, I introduced, in the form of an appendix, a sketch of the various notions which had been advanced respecting the ordinary motions and sounds of the heart; and by comparing them with well-established physiological or pathological facts, I was enabled to set aside as untenable the greater number of these views, and to state with some precision the weak or uncertain points of those which still deserved consideration and experimental investigation. As this sketch will still serve to illustrate the history of the subject, I introduce it in a note below;[*] and it remains for me here to cite from the last edition of this work the state in which I found the subject when I undertook to investigate it by experiments. The text is reprinted without alteration from the third edition, 1835, with the exception of the notes enclosed in brackets.

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[*] Others, besides the authors quoted, may have supported the following views on the subject in question; but it is only the views which I wish to deal with, and I name the writers only to show that the arguments which each has advanced have been carefully studied.

1. M. Laennec. *a.* 1st sound, impulse, and pulse, caused by the ventricular systole. *b.* 2nd sound by the systole of the auricles.—*Remarks.* *a.* Generally admitted and proved by various facts and experiments. *b.* Disproved by the fact noticed by Harvey and Haller, and confirmed by modern experiments, that the auricular contraction immediately precedes that of the ventricles: also by this fact, that both sounds sometimes continue after the auricles have ceased to contract.[*]

[*] Dr. Hope's Experiments on Asses. See his Work, p. 36.

From the statements below it may be considered as demonstrated by former experiments:—

2. Mr. Turner.[*] 2nd sound produced by the falling back of the heart on the pericardium after the systole of the ventricles.—*Remark.* Disproved by the fact that the sound continues when the heart pulsates out of the pericardium.

3. Dr. Corrigan.[†] *a.* Impulse and 1st sound caused by the rush of blood into the ventricles during the auricular systole. *b.* 2nd sound by the ventricular systole, which he considers to be instantaneous.—*Remarks.* *a.* Disproved by the clearly ascertained facts that the 1st sound and impulse accompany the systole of the ventricles when the auricles have ceased to contract. *b.* Disproved clearly in large animals by the ventricular systole, (which is not instantaneous) and the pulse of arteries near the heart, evidently preceding the 2nd sound;[‡] and further disproved by several pathological phenomena.

4. Dr. David Williams.[§] 2nd sound caused by the flapping open of the auriculo-ventricular valves against the sides of the ventricles; these valves he supposes to be opened by the musculi papillares.—*Remark.* This is contrary to the received opinion of anatomists with respect to the functions of the auricular valves and musculi papillares, and there is no collateral argument to maintain so gratuitous an assumption.

5. M. Pigeaux.[||] *a.* 1st sound produced by the blood rushing into the ventricles at the moment of their diastole. *b.* 2nd sound by the collision of the blood against the walls of the aorta and pulmonary artery. *c.* The ventricles contract in a moment of silence before the second sound. *d.* The intensity of the sounds proportioned to the force by which the blood is impelled.—*Remarks.* *a.* Opposed by the facts stated against 3 *a;* opposed also by many pathological facts, such as the occurrence of a murmur with the 1st sound, in case of diseased semi-lunar valves. *b.* Disproved by the fact that the 2nd sound occurs distinctly *after* the pulse in the carotids, and therefore after that in the larger arteries. *c.* Opposed by the observation that the 1st sound and ventricular systole occur together and correspond in duration. *d.* This is opposed by the morbid phenomena of dilatation of the ventricles, which always increases the first sound, and of hypertrophy, which diminishes both sounds.

6. M. Majendie.[¶] 1st sound and 1st impulse produced by the ventricular systole impelling the apex with a shock against the walls of the chest between the fifth and sixth ribs; the 2nd sound and 2nd impulse by the blood, in refilling the ventricles at their diastole, forcing their parietes with a shock against the sternum.—*Remarks.* Completely disproved by the fact that both sounds continue when the heart does not touch the parietes of the chest.[**]

7. M. Rouanet.[††] *a.* 1st sound caused by the closing of the mitral and tricuspid valves against the auriculo-ventricular orifices during the ventricular systole. *b.* 2nd sound by the reaction of the blood in the arteries on the semilunar valves at the moment of the ventricular diastole.

8. Mr. H. Carlile.[‡‡] *a.* 1st sound produced by the rush of blood into the arte-

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[*] Med. Chir. Trans. Edin. vol. iii.

[†] Trans. of King's and Queen's Coll. of Phys. Ireland. [Dr. Corrigan has now abandoned this view.]

[‡] Dr. Hope's Experiments, p. 31 of his work; and those of Mr. Carlile, Dublin Journal of Medical Science, vol. iv.

[§] Edin. Med. and Surg. Journal, Oct. 1829.

[||] Arch. Générales de Médecine, Juillet et Novembre. 1832.

[¶] In Lectures delivered at the College of France, in 1824, reported in the Lancet, Feb. 1835.

[**] See the Experiments of Dr. Hope, (p. 30 et seq. of his work,) of M. Bouillaud (Journ. Hebdom.) and my own, as described further on in the text.

[††] Journ. Hebdom. No. 97: also Mr. Bryan, Lancet, Sept. 1833; and M. Bouillaud, Journal Hebdomad. 1834. [This view was proposed by Dr. Billing, in Feb. 1832; but as far as regards the 2nd sound he was anticipated by Dr. Carswell, whose opinion was publicly announced in the Academy of Medicine at Paris, in July 1831. See Dr. Joy's able and impartial article on Diseases of the Organs of Circulation, Library of Medicine, vol. iii. p. 262..]

[‡‡] Dublin Journal of Medical Science, vol. iv. The essay was likewise read at the Cambridge Meeting of the British Association.

1st. That the auricles contract first, producing no sound.
2nd. That the auricular contraction is immediately fol-

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ries during the ventricular systole. *b.* 2nd sound by the reaction on the semilunar valves, as stated in *b.* 7.

9. Dr. Hope. *a.* 1st sound and impulse, caused by the ventricular systole. *b.* 2nd sound and *back stroke,* or 2nd impulse, by the ventricular diastole. The natural as well as morbid sounds produced by the motions of the contained fluid.

Before we sift the questionable points in these three last views, it will be proper to review the principal grounds on which we adopt their description of the sounds and motions, in defiance of many preceding authorities. Having been present at some of Dr. Hope's experiments on the ass, I had ample opportunity of convincing myself that the sounds were connected with the motions of the ventricles only. When the pericardium was laid open, and the large heart exposed, vigorously pulsating; the eye watching it, the hand grasping it, and the stethoscope applied to it, gave perfectly corresponding impressions; insomuch, that on substituting touch for hearing, it was difficult to banish the impression that one still *heard* the double sound which was so exactly represented in quality and duration by the motions of the ventricles, as felt and seen; and on combining touch and hearing, by applying the hand and the stethoscope at the same time, these impressions, which corresponded in nature and duration, were found also to be perfectly simultaneous. The apex of the heart was observed and felt to strike against the ribs at each systole, and thus was explained the impulse. The motions of the auricles, when regular, preceded the ventricular motions and sounds; they were slight and undulatory, increasing from the sinus to the appendix, where they terminated suddenly, and were immediately followed by the ventricular systole. They afterwards became irregular, sometimes failing and sometimes occurring twice slightly during the period of ventricular repose, and in one experiment entirely ceased some minutes before the movements and sounds of the ventricles. In no instance were they attended with any perceptible sound. This account is confirmed by the experiments of Mr. Carlile, which satisfactorily explain the succession of the motions of the auricles and ventricles; but they were performed on animals too small to illustrate the sounds. He very justly shows that the pulse cannot be simultaneous in all the arteries at once, but must be successive, transmitted in a wave from the heart to the end of these elastic tubes.

Although it seems fairly established that the first, or dull sound, is produced by the systole of the ventricles; and the second, or quick one, by their diastole, it is by no means clearly explained in what way these actions generate these sounds. The following causes have been severally assigned as physically capable of generating the first sounds during the systole of the ventricles. 1. The collision of the particles of fluid in the ventricles. (Dr. Hope, Dr. Spittal.) 2. The rush of blood into the great arteries. (Mr. Carlile.)—3. The closing of the mitral and tricuspid valves. (M. Rouanet, Mr. Bryan.)—4. The muscular contraction itself.

The first of these explanations is ingeniously proposed by Dr. Hope, but he advances no facts in direct proof of the hypothesis. In a number of experiments which I have made on the generation of sound, I have found liquids, of all bodies, the most difficult to excite to sonorous vibration; and although they readily transmit vibrations already produced in solids, it requires a combination of circumstances to make them originate sound. This is consistent with the explanation which I have given of the production of sound, (p. 5, *et seq.*) for impulses which throw solids into sonorous vibrations, are expended in liquids in causing a displacement of their particles. On making an experiment with a gum elastic bottle, by filling it with water, and then forcibly compressing it under water by the end of the stethoscope, (avoiding the use of the hand, for that produces its own muscular sound,) I have failed in procuring any sound at all approaching to that of the heart's contraction. The blood yields readily to the contracting ventricle, and there being no obstacle to the escape of blood from it, further than the weight of the arterial column, which the normal action of the heart can quietly and steadily lowed by the ventricular systole, which is accompanied by the first or dull sound. This systole, by straightening the

overcome, it passes into the arteries without vibration. But if there be an obstacle to the current of the blood from the ventricle, whether that obstacle be a narrowing or a projection in the orifice, the current will act on it just as the bow does on the string of a violin ; a sound will be excited, and thus are produced valvular murmurs. Again, if, instead of the orifices being narrowed, the heart contracts with unnatural briskness, expelling its contents with convulsive energy, the natural outlets then become relatively narrow, and are thrown into vibrations ; this is the *rationale* of the bellows murmur which accompanies the jerking pulse of pericarditis and the irritation of inanition. But the difference of these sounds, and of the circumstances that excite them, from those of the normal action of the heart, makes me hesitate to refer the latter to the same principle ; and the fact that the morbid are often superadded to the natural sounds, also inclines me to think that they have a distinct cause.

2. The second explanation of the first sound, the rush of blood into the larger arteries, is perhaps less liable to the acoustic objection before urged, than the preceding opinion, for the blood has acquired an impulse when it enters the arteries, and if its course there is not free, it might readily produce a sound. But, in their natural state, the arteries give passage to the blood as smoothly as the heart parts with it, and it would prove an imperfection in nature were it otherwise. Moreover, if this explanation were true, the large arteries rather than the heart would be the principal seat of the sound ; and the sound should be increased by an hypertrophied heart, with a strong pulse, and diminished by a dilated heart and a weak pulse ; yet the reverse of these is presented in nature.

3. The closing of the auricular valves. The principal objection to this as the only cause of the first sound, is, that it must be instantaneous, and confined to the first part of the ventricular systole ; whereas we know that the first sound is prolonged during the whole period of this action.

4. Although Laennec referred the first sound to the systole of the ventricles, he did not attempt to define the physical cause of its production. In the former edition of this work, I ventured to class it among the muscular sounds which Dr. Wollaston* first noticed to occur in all cases of rapid muscular contraction. This sound may be exemplified by applying the fleshy part of the thumb to the stethoscope or naked ear, and bending and straightening the thumb. It is louder in muscles that are thin, and in a state of considerable tension ; and it is remarkable that it does not cease with the apparent movement, but continues as long as the muscle remains contracted and tense ; it then takes on an intermitting character, like the noise of the rolling of a carriage over rough pavement, whence Dr. Wollaston was led to infer that muscular action is not perfectly continued, but consists of a series of minute contractions and relaxations. A good example of it may be obtained on applying the stethoscope to the neck of a person who holds his head back towards the opposite side, and then throws the platysma myoides into contraction. It still appears to me that the most simple and satisfactory way of accounting for the first or systolic sound of the heart is to refer it to this class of sounds. Their physical production seems to depend on the sudden tightness or tension into which the fibres of muscles are thrown when they contract ; and the self-acting power of these fibres constitutes them the motors as well as the subjects of sonorous vibrations. Here we have to remark the extreme facility with which the motions of solids produce sounds, compared with those of fluids ; for it is almost impossible to touch, stretch, bend, or compress solids, without throwing them into sonorous vibrations. In this case we have a series of cords or fibres brought by a zig-zag tension, into a state of rigidity capable of sonorous vibrations, and the impulses developing these vibrations are

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* Croonian Lecture, Phil. Trans. 1810.

anterior convexity of the ventricles, brings their apex into forcible contact with the ribs, and thus is produced the im-

communicated by the oscillations of those contracting fibres themselves; these oscillations, in a greatly magnified degree, are apparent in the quivering seen and felt in the muscles of a horse drawing with unusual effort. The varieties observed in the contraction of the heart seem to me to be perfectly explicable on this principle. The sound begins the moment the fibres arrive at a state of tightening or tension; it continues until the contraction is completed and the blood expelled from the ventricle, and ceases the instant of the diastole. To perceive more readily the effect of hypertrophy, and of dilatation, let us attend to the sounds produced by the tension of linen or canvass, (for muscle is, mechanically speaking, equally a web of fibres,) and we shall find that in proportion as we thicken the substance, we obscure the sound which is produced on briskly stretching it; but when we use thin and simple webs, the sound becomes proportionally loud and clear. I shall not pursue the illustration of this explanation further, for I introduce it here only interrogatively, as deserving a place among other views, on the claims of which, future observation and experiment must decide. I must only remark, that M. Pigeaux is in error when he maintains that muscular sounds cannot be produced under water: I find them more distinct and free from adventitious sounds of the surface, and I have been able to imitate the sounds of the heart very exactly by muscular movements of the hand under water. I will conclude with the question, if the first sound of the heart is produced by another cause, what becomes of the muscular sound in this case of rapid muscular contraction?

We now come to the subject of the second sound, which, although certainly occurring at the moment of the diastole of the ventricles, has received several different explanations as to its physical cause. The only two which appear tenable in the present state of our knowledge are—1. The reaction of the arterial columns of blood against the semilunar valves. 2. The impulse of the blood from the auricles refilling the ventricle at its diastole.

1. The first of these bears a very inviting aspect, for the 2nd sound is just of that abrupt flapping character that might be supposed to result from the action of a thin valve. According to this view, the arteries, more than the heart, should be the seat of this sound. The tense column which throws these valves into play, should receive their shock more forcibly than the heart, which at that moment has become flaccid, and ill adapted to transmit sound or impulse (backstroke) through the whole of its substance. There are some cases of disease [in Dr. Hope's work] which seem to militate against this view; but as the history of these cases was not studied with especial reference to this point, their accuracy is not, perhaps, clear enough to warrant our arguing upon them; I therefore still do not consider this view as disproved, and it should claim attention in future investigations.

2. This is Dr. Hope's explanation of the second sound: when the diastole takes place, the blood, impelled by a number of concurrent circumstances, shoots with instantaneous velocity from the auricles into the ventricles; and the reaction of the ventricular walls on its particles, when their course is abruptly arrested by the completion of the diastole, is, he conceives, the cause of the loud, brief, and clear sound. The concurrent circumstances which impel the blood into ventricles at the moment of their diastole, are the distension of the auricles in which the blood has been accumulating during the ventricular contraction; the weight of the ventricles collapsing on the auricles thus distended; the width of the auriculo-ventricular orifices; and lastly, the sucking power of the ventricle in its diastole. With respect to this last, Dr. Hope does not assume that the ventricles have an actively dilating power further than what proceeds from the physical elasticity of their parietes, but such a power has been ascribed to them by Bichat, Pechlin, Carson, and others, and even by Laennec; and although opposed to what we at present know of animal dynamics, it would be rash to absolutely deny

pulse. This systole, by throwing an additional quantity of blood into the arteries, causes the arterial pulse which in arteries near the heart is synchronous with the ventricular systole; but in those more distant succeeds it at an interval occupied by the transmission of the wave through the blood along the elastic tubes from the heart.

3rd. That the ventricular systole is immediately followed by the diastole, which is accompanied by the 2nd or short sound.

4th. That there is then an interval of rest, at the conclusion of which the auricles contract, and the series of motions is repeated as before.

But it was still uncertain in what way the ventricular systole produced the first sound, or how the diastole developed the second; and various opinions were broached by different writers on these subjects. Thus the first sound was ascribed by Mr. Carlile to the rush of blood into the great arteries; by M. Rouanet and others to the closing of the auriculo-ventricular valves; by Dr. Hope to the collision of the particles of fluid in the ventricles; and, by myself, to the muscular contraction itself.

The 2nd or short sound, accompanying the diastole of the ventricles, was ascribed—by Dr. Hope, to the impulse of the blood from the auricles refilling the ventricles;—by M. Rouanet, Mr. Carlile, M. Bouillaud and others, to the reaction of the arterial columns of blood against the semilunar valves.

I concluded the appendix before quoted, by recommending Dr. Hope to investigate experimentally these points, which were there shown to be doubtful; but his engagements, and other circumstances, having prevented him from doing so, I lately undertook the task. I made a point of ensuring Dr. Hope's presence and testimony at the experiments, and I

the possibility of its existence. I would suggest that the injection of the coronary arteries, which occurs the instant the systolic action ceases, may somewhat contribute to the dilatation of the ventricles. Whatever be the cause, the diastole in large animals is sufficient to force open the hand of a person grasping the ventricles, and it is therefore not surprising that this should have been ascribed to an actively dilating power. It is in favour of Dr. Hope's explanation of the second sound, that it does not falsify Laennec's signs of disease of the auricular valves; and although, for acoustic reasons before stated, I would have placed the seat of the sound in the parietes of the ventricles rendered momentarily tense by the sudden influx of blood, rather than in the motions of the fluid, I have until lately [in deference to Dr. Hope's statements] inclined to this explanation of the cause of the second sound. It needed, however, as Dr. Copland observes, further confirmation; and I would add, that the whole subject of the sounds of the heart required further research.

conferred with him and several other gentlemen as to the best mode of performing them.*

Through the kind assistance of Sir Benjamin Brodie, in providing me with some of the woorara poison, the results were more satisfactory, whilst the animals were put to less suffering, than in any prior experiments. This poison, better than any other, suspends the action of the nervous system without affecting the irritability of the heart, and it proves fatal by destroying the animal sensibility on which the act of respiration depends. If the respiration be then maintained artificially, the heart will continue to beat, and the circulation will proceed for a considerable period ; until, in fact, the various organs become cooled and otherwise injured, for want of the continuance of other processes, which are in some way maintained by the nervous influence. Thus in the following experiments the heart continued to act vigorously for more than an hour after the extinction of animal life ; and might have done so longer, had it been necessary ; but all the results were clearly ascertained and confirmed by those present within that period.

Experiment I.

About twenty grains of woorara, moistened with water, having been inserted in an incision in the haunch of a donkey two months old, the animal died in fifteen minutes. Artificial respiration was immediately established, and the chest being opened, the pericardium was slit down and the heart exposed. Its pulsations were regular and vigorous, the auricles contracting immediately before the ventricles. The double sound was distinctly isochronous with the systole and diastole of the ventricles. The following points were then observed and noted, after repeated examinations by several present.†

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* I was present at an experiment attempted by Dr. Hope, in November last, at Mr. Field's ; and he then planned modes of suspending the action of the valves, similar to some of those afterwards adopted in my experiments. The animal was killed by a blow on the head, and this so much impaired the heart's action, that no results were obtained. [In this note I gave Dr. Hope credit for more than was strictly due to him, because I was resolved that he should have no just cause for complaint : even this concession has not satisfied him, and I must therefore state, in justice to myself, that even his *plans* of experiment were devised only after much conference with myself, and long after he had perused my printed summary, given in the preceding pages.]

† The following gentlemen were present ; and I here beg to offer to them, and to those who assisted at the other experiments, my thanks for their kind co-operation and testimony :—Dr. Arnott, Author of the Elements of Physics, &c. ;

Observation 1. The 1st sound was equally audible on all parts of the ventricles.

Obs. 2. The 2nd sound was most distinct near the roots of the great arteries, being audible there in the weaker pulsations, when it could not be heard by the stethoscope applied to other parts of the ventricles.

Obs. 3. Pressure on the arterial roots by the fingers, or by the stethoscope, invariably stopped the 2nd sound. Slight pressure caused a whizzing, or bellows murmur, with the 1st sound.

Obs. 4. On pushing the auricles, by the end of a finger, into each auriculo-ventricular opening, the ventricular contractions became weak and irregular; but the 1st sound, although weak, was still heard alone.

Obs. 5. At each systole, the sudden tension or tightening of the ventricles was felt, by the finger applied to the body, as an abrupt shock, with which the 1st sound exactly coincided.*

Obs. 6. The left auricle was cut open, and the mitral valve partially destroyed; the blood issued in jets at each ventricular systole; yet the 1st sound still accompanied the systole. The 2nd sound was not heard after this incision.†

Obs. 7. The right auricle was also freely laid open; still the 1st sound continued.

Obs. 8. I pushed my finger through the mitral orifice into the left ventricle, and pressed on the right, so as to prevent the influx of blood into either ventricle: the ventricles continued to contract strongly, (especially when irritated by the nail of the finger in the left,) and the 1st sound was still

Mr. Babington, Surgeon to St. George's Hospital, &c.; Mr. Good, Surgeon to St George's and St. James's Dispensary; Dr. Hope, Assistant Physician to St. George's Hospital, &c.; Mr. Henry Johnson, Demonstrator of Anatomy, &c.; Dr. T. Peregrine, House Surgeon at St. George's Hospital; Mr. G. Smith, Lecturer on Anatomy, &c.; Mr. Tatum, Lecturer on Anatomy, &c.

To Mr. Tatum, Mr. Henry Johnson, and Mr. H. James Johnson, I am especially obliged, not only for their able assistance, but also for the use of their splendid new dissecting-room, and its commodious appurtenances, in Kinnerton-street, where these experiments were performed.

* This Observation was, I think, suggested by Dr. Hope. [Be it observed, that I have carefully acknowledged every point in which Dr. Hope assisted in the experiments. In justice to those gentlemen, I should state that I received quite as much assistance from Mr. Good and Mr. Henry Charles Johnson, one of whom was present the whole time, and during a part of the experiments when Dr. Hope was absent.]

† The results after 6 were witnessed by Dr. Hope, Mr. H. Johnson, and myself, the other gentlemen having left. The observations were noted during or immediately after the experiment; generally by Dr. Hope or myself.

distinct, but not so clear as when the ventricles contracted on their blood.

Obs. 9. The same phenomena were observed when both the arteries were severed from the heart.

Until the auricles were cut open, as mentioned in 6 and 7,) the 2nd sound was audible in all the strong pulses of the heart; but it was not heard after, although upwards of 30 pulses, most of them vigorous, took place. Ten or twelve strong contractions occurred after the introduction of the finger, as mentioned in Obs. 8.

This experiment lasted an hour and twenty minutes from the commencement of artificial respiration.

Experiment II.

About fifteen grains of woorara (powdered, and made into a paste with water) were introduced into a wound under the haunch of a young ass, about six weeks old. The animal died in about thirty-five minutes. Artificial respiration was then immediately established, and the chest opened by cutting through the cartilages of the ribs, to the left of the sternum, and along the upper margin of one of the ribs near the shoulder, and then breaking back three or four ribs, so as to expose the contents of the left side of the chest. The following points were noted, as observed by several present.*

Obs. 1. Before the pericardium was opened, the 1st and 2nd sounds were very distinctly heard, although the heart touched no part of the parietes of the chest.

Obs. 2. Both sounds were distinctly heard through a lobe of the lung interposed between the heart and the stethoscope.

Obs. 3. The pericardium being completely slit open, the 2nd sound was observed to be, decidedly, most distinct at the origin of the pulmonary artery and aorta, where it was louder than the 1st sound, and had perfectly its natural short, clear, flapping character. With the stethoscope applied on the body of the ventricles, the 2nd sound was heard less distinctly, and seemed more obtuse and distant.

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* Mr. Bushell, Lecturer on Anatomy, &c.; Mr. Good, Surgeon to St. George's Dispensary, &c.; Dr. Hope; Mr. H. Johnson, Surgeon, &c.; Mr. Keate, Surgeon to their Majesties, &c.; Dr. Macleod, Physician to St. George's Hospital, &c.; Dr. Page; Mr. Partridge, Junior Professor of Anatomy at King's College, &c.; Mr. Malton and Mr. Seagram, Pupils at St. George's Hospital; Mr. Willesford, Surgeon, &c.

Obs. 4. When the stethoscope was applied to the aorta about three inches from its origin, the 2nd sound (without the 1st) was heard following the systole of the ventricles as felt by the observer's finger.*

Obs. 5. The aorta and pulmonary artery being for a few seconds compressed between the finger and thumb, the 1st sound was accompanied with a bellows murmur, and the second sound ceased, during the continuance of the compression. This experiment was repeated several times by Dr. Hope and myself.

Obs. 6. A common dissecting-hook was passed into the pulmonary artery, and was made to draw back and thus prevent the closure of the semilunar valves: the 2nd sound was evidently weakened, and a hissing murmur accompanied it. A shoemaker's curved awl was then passed into the aorta, so as to act in the same way on the aortic valves: *the 2nd sound now entirely ceased, and was replaced by a hissing.*

Obs. 7. The hook and the awl were withdrawn; *the 2nd sound returned, and the hissing ceased.* This and the preceding experiment were repeated, and observed by Dr. Hope, Mr. H. Johnson, Mr. Malton, and myself.

Obs. 8. Experiment 6 was repeated, with the same result, and whilst Dr. Hope listened, I withdrew the awl from the aorta. He immediately said, "Now I hear the second sound." I then removed the hook from the pulmonary artery; Dr. H. said, "Now the second sound is stronger, and the murmur has ceased."

Obs. 9. The pulmonary artery was cut open, and the finger introduced into the right ventricle;—the heart continued to contract irregularly, and the 1st sound alone was obscurely audible.

Obs. 10. Slight contractions took place after the ventricles were laid open, and the columnæ carneæ were seen to contract simultaneously with the fibres of the ventricles.

These observations lasted during an hour and ten minutes after the commencement of artificial respiration; and until the opening of the artery in Obs. 9, the contractions of the heart were generally regular and vigorous.†

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* This observation was suggested by Mr. Keate, and Obs. 2 by Dr. Hope.

† Two other asses were subjected to experiment, but, from accidental circumstances, no conclusive results were obtained from them. One was in so weakly a state before the poison was applied, that when the chest was opened, the heart pulsated very feebly, and without any sound. A curious circumstance was noted in this case: when the ventricles had almost ceased to move, the auricles went on contracting regularly and fully, but not simultaneously, for the movement of the

The deductions from these results are simple and obvious; and they not only decide between the views before stated, but they appear to me to demonstrate more satisfactorily the true seats and causes of the heart.

That the first sound is not caused by the rush of blood into the great arteries, (as supposed by Mr. Carlile,) is proved by Obs. 4, 6, 7, 8 and 9, of Exper. I.; and Obs. 9, of Exper. II.; in which the first sound continued, although little, and in the latter cases no blood could have been thrown into the arteries. A further proof against this view may be seen in Exper. II. Obs. 4, from which it appears that the first sound is much less audible in the large arteries than in the heart.

That the first sound is not dependent solely on the closing of the auriculo-ventricular valves, (as imagined by M. Rouanet and others,) is evident from Exper. I. Obs. 4, 6, 7, 8, 9, in which the closure of these valves was partially or completely prevented, yet the 1st sound still continued.

That the first sound is not produced by the collision of the particles of fluid in the ventricles, (as formerly conceived by Dr. Hope,) appears from Obs. 4, 8 and 9, of Exper. I.; and Obs. 9, of Exper. II., in which the sound was produced, although there was no blood in the ventricles.

That the first sound is produced by the muscular contraction itself, may be considered as proved by Obs. 8 and 9 of Exp. I.; in which every other possible source of sound was excluded, and the 1st sound still accompanied the systolic action of the ventricles.*

That the second sound is produced by the reaction of the ar-

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right preceded that of the left by about the same interval as the auricular movements generally precede the contraction of the ventricles; and it appeared that these movements of the auricles were sufficient to maintain to a certain degree the circulation, for arteries, when wounded, yielded florid blood, but not in jets.

In the other experiment, the substance of the ventricles was accidentally wounded in opening the chest; this occasioned much hæmorrhage from the coronary artery, and weakened the heart's action so much, that the second sound was not heard. This result was generally remarked, that whenever the ventricular systole was weak, and the arterial tension was not kept up by it, the second sound ceased. These unsuccessful trials were useful to us, in familiarizing us with the anatomy, and in preparing for the others.

* This view of the cause of the 1st sound was first published by me in the first edition of this work in 1828, and I am not aware that it has been entertained by any other writer. [More recently M. D'Espine and others have also advanced this view.] M. Majendie is in error when he ascribes a similar opinion to Laennec, who, on the contrary, only associated the abnormal murmurs with the muscular sound produced by a fancied spasm of the heart and arteries; but neither in his works, nor in his lectures, did he give any opinion as to the physical cause of the healthy sounds of the heart.

terial columns of blood tightening the semilunar valves at the ventricular diastole, is clearly proved, not only by the situation of these valves being the especial seat of the sound, (Exp. I. Obs. 2; and Exp. II. Obs. 3, 4,) but also by the numerous observations in which the cessation or reproduction of the sound was effected by the suspension or restoration of the action of these valves. (Exp. I. Obs. 3; Exp. II. Obs. 5, 6, 7, 8.)

It being thus proved that the 1st sound is essentially produced by the tightening of the muscular parietes of the ventricles, and the 2nd sound by the subsequent sudden tension of the semilunar valves, it is easy to perceive how various circumstances may increase or diminish the sounds, as they augment or impair the degree or abruptness of this tightening or tension in these parts. Thus the mass of blood in the heart increases the clearness of the 1st sound, by affording an object, around which the fibres effectually tighten; whilst the auricular valves, by preventing the reflux of this blood, increase its resistance, and thus add to the tension necessary for its expulsion. Probably, in common pulsations, the ventricles do not attain the degree of tension which is sonorous until the closing of the auricular valves: this closure, as the commencement of the resistance, brings at once to its acmé the muscular tension, which continues until the contents of the ventricles are sufficiently expelled. This accounts for the sudden or flapping commencement often perceptible in the 1st sound, and it suggests how the due action of the auricular valves generally contributes to its clearness. The degree and abruptness of the systolic tension was well seen in Obs. 5, of Exp. I.; and the external feel and view of the systole gave us the impression of its being a motion in itself sufficient to produce sound. The auricular valves, the cordæ tendineæ, the columnæ carneæ, and internal fibres of the ventricles, if they attain the same degree of tension as the exterior of the ventricles, may have an equal share in the production of the 1st sound;* but I am disposed to think, that what we hear proceeds chiefly from the contracting tenseness of the external walls of the heart, both because they are nearer to the ear, and because in Exp. I. Obs. 8, the contraction of the left ventricle upon my finger within it, was by no means so

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[* This passage shows that I did not deny that the tightening of the auricular valves is an auxiliary cause; these experiments were not conclusive on this point. Subsequent experiments have convinced me that these valves are the chief seat of the flapping commencement of the first sound, but not of its most characteristic and essential part.]

abrupt or strong as that of the exterior, as felt by the other hand, and still heard through the stethoscope.

The termination of the systole of the ventricles is abrupt, being immediately followed by the diastole: and it is obvious that the first effect of this must be, to occasion the sudden closing of the semilunar valves, pressed now only on their concave side by the force of the arterial column of blood. Hence the 2nd sound immediately succeeds the 1st, or rather appears to terminate it, by its abrupt, clear *flap*, which in the healthy heart differs as much in character from the 1st sound, as the simple valves by which it is produced differ from the thicker muscular web of the ventricles, the tightening of which causes the 1st sound. The mobile state of these valves, the bulk of blood propelled by the ventricular systole into the arterial column, and the suddenness of the diastole by which this column is brought to press fully backwards on the valves, are the circumstances which give clearness and loudness to the 2nd sound; and it may be seen, in the foregoing experimental observations, how various causes interfering with these, impaired or suspended the 2nd sound. (Exp. I. Obs. 3, 4, 6, &c., Exp. II. Obs. 5, 6, 7, 8, 9.)

The whole of these observations abound in facts which concur with many former experiments in entirely overthrowing the extraordinary opinions of M. Majendie on the sounds of the heart, which I have noticed in a former page. For the satisfaction of some of those present, whose doubts had been raised by the positive tone of the assertions of so distinguished a physiologist, and by the plausibility of some of his explanations, Obs. 1 and 2 of Experiment II. were made, and all the gentlemen present agreed that the same sounds that had been heard before the chest was opened, were most distinctly produced after that operation, when the heart struck against nothing in its motions. After this, it is quite unnecessary to go through the details of the ingenious superstructure which M. Majendie has erected on a foundation thus proved to be erroneous. It is surprising that such an eminent observer should permit himself to be so blinded by a favourite notion, as to strain conclusions in its favour, and tax his ingenuity for its support, in total disregard of simple and decisive facts. On reading attentively the various arguments and cases which M. Majendie brings forward, (as reported in the Lancet, Feb. 1835, from his Lectures) I find there scarcely any phenomena clearly described which will not admit of a more simple and satisfactory explanation from the views which have been established by the preceding experiments. Certain of his assertions, such as that water or air in the pericardium entirely suspend the sounds of the heart, will be viewed by experienced auscultators rather as proofs of an imperfection in the observer's hearing, than as arguments in support of his opinions.

Although it is proved by the preceding observations that the impulse of the heart against the chest does not produce its natural sounds, yet I am disposed to admit, that in violent action of the organ, its more sudden and abrupt strokes against the chest do cause a sound, which constitutes the loud termination of the 1st sound in these cases, and which seems nearer the ear, and more like a knock, than what is heard in the ordinary action of the heart. In common pulsations, the apex of the organ is drawn upwards and forwards at each systole, and sliding obliquely on the smooth pericardium, does not impel against the ribs with sufficient abruptness to cause sound. But in quicker and more violent pulses, the abruptness of the motion, and the force of the blow against the side of the chest, are such as can scarcely fail to produce sound.*

Another circumstance which seems to have misled M. Majendie, is the fact that the sounds of the heart are louder when the chest is entire, than when the organ is exposed. This, instead of proving that the sounds are necessarily produced on the thoracic walls, merely exemplifies the general fact, that the sounds of a sonorous solid are increased by being confined in a close cavity with elastic walls, which augment the sound after the manner of a sounding board.

[Such were my conclusions after my experiments in 1835; and it is very satisfactory to me to be able to say that now, after many more experiments performed by myself and others, and after five years' continual attention to the healthy and morbid phenomena of the heart, my opinions have been

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[* Dr. Hope has urged against this conclusion an argument first used by Mr. Bryan, which is more specious than real. Mr. B. asserts that the heart is kept in its place by atmospheric pressure, and cannot recede so as to knock against the walls of the chest, without overcoming that pressure, which amounts to 15lbs. to the square inch. This is a strange error: the heart is not kept in its place by atmospheric pressure; for the lung yields readily before, behind, and above it, and it is free to move from or to the walls of the chest by displacing or compressing the unresisting tissue of the lung. Thus, as stated in the text, in certain postures the heart hangs near enough to the walls of the chest to strike them at each pulse. In other postures, or with full inspiration, the lung intervenes, and the heart does not so strike.]

changed in no material point. It is very gratifying to me to be able to add, that many distinguished writers have adopted these conclusions.

I have now to perform a less agreeable duty in connexion with these experiments—to substantiate my claim to them. I would willingly waive this, and forego all credit connected with the subject; for it is a theme which must remind others, as well as myself, of a friendship painfully blighted; but misrepresentations, recently published, compel me, for truth's sake, to speak out. In the following statement of facts and dates, every assertion is substantiated by reference either to published works, living witnesses, or to autograph letters left with the publisher for inspection. I refer to no written documents of my own, because, in a question like this, the unsupported testimony of the contending parties ought not to be received.

Dec. 1831. Dr. Hope's first edition appeared, with the account of his experiments and conclusions.

Jan. 1832. Dr. Williams reviewed Dr. Hope's work in the London Medical and Physical Journal; and pointed out defective parts in the experimental inquiry which needed further investigation.

Sept. 1833. Dr. W. published an appendix to the second edition of his work, in which the various views on the motions and sounds of the heart were discussed, the defective points of these views stated, and subjects for further experimental investigation suggested.

Sept. 1834. Dr. W. proceeded to Edinburgh during the meeting of the British Association, with the intention of performing experiments on the debated points: this intention was made known to Dr. Arnott, Dr. Alison, Dr. Sharpey, Dr. Spittal, and others. The experiments were not performed, because animals could not be procured for the purpose.

Nov. 1834. The last of Dr. Hope's articles (Diseases of the Valves) appeared in the Cyclopædia of Practical Medicine. In this were re-asserted, without expression of doubt, the same views which had appeared in his work.

Nov. 1834. Dr. Hope attempted to perform experiments on the points previously suggested by Dr. W., and proposed certain modes of manipulating in these experiments, Dr. T. Davies, Mr. J. H. Johnson, and Dr. Williams, being present. This experiment failed, as stated in note, p. 300.

Jan. 1835. Dr. Williams being about to publish a third edition of this work, and unwilling to leave the matter undecided, again undertook to perform the experiments in question. He applied to the late Mr. Field to assist him, (a letter to this effect is at the publisher's,) was favoured with some woorara from Sir B. Brodie, and requested the use of the dissecting-room in Kinnerton-street, and engaged the assistance of the gentlemen teaching in that school, and of others, Dr. Hope amongst them.

Feb. 1835. Dr. W. procured animals for the experiments, and, assisted by the gentlemen alluded to, performed them; undertook the whole management of them, and paid all the expenses connected with them.

See Sir B. Brodie's letter, printed below. Letters from the gentlemen who chiefly assisted, and a statement by the porter of the dissecting-room, are at the publisher's, further to substantiate these statements.

It was not until Dr. W.'s account of these experiments was actually in the press, that Dr. Hope claimed a share in them, and disputed Dr. W.'s right to publish them. The question was referred to Sir Benjamin Brodie for arbitration: the following is his answer:

"14, *Saville-row*, *March* 10, 1835.

"MY DEAR SIR,

"I understand from yours and Dr. Hope's statements—

"1st. That you and Dr. Hope have both been engaged for a considerable time in researches respecting the pathology of the heart:

"2ndly. That Dr. Hope formerly instituted experiments with a view to illustrate this subject, at which he invited you to be present:

"3rdly. That since then you and Dr. Hope have been in the habit of discussing questions arising out of these experiments, and that you contemplated making others conjointly:

"4thly. That you frequently urged Dr. Hope to proceed with the projected experiments, but that his various engagements prevented his doing so:

"5thly. That at last you applied to Mr. Tatum for the use of the new dissecting-room; procured animals for the purpose of the experiments; asked several gentlemen to assist you in making them; and invited Dr. Hope to them also.

"In addition to all this, I am informed by some of the gentlemen who assisted you, that the experiments were made almost entirely under your direction.

"Now, if these statements be correct, I own that I do not see that Dr. Hope can well complain of your making use of the experiments in the third edition of your work now in the press; at the same time I am of opinion that, in doing so, you should be careful to explain what share Dr. Hope had in projecting and planning the experiments in the first instance, and that you should acknowledge whatever assistance you derived from his suggestions at the time of the experiments being made.]

"I am, dear Sir,
"Always yours truly,
"B. C. Brodie.
"Dr. Williams."

For explanation of the origin of Dr. Hope's desire to appropriate these experiments, I can only refer the reader to his works published before their performance and those published since. The conclusions of the latter are so different from those expressed in the former, that it would have been humiliating to acknowledge that the correction originated with another person. I forbear to state the course which he has pursued. His own account is contained in the last edition of his work.]

II.—FIRST REPORT

OF THE

LONDON COMMITTEE OF THE BRITISH SCIENTIFIC ASSOCIATION,

ON THE

MOTIONS AND SOUNDS OF THE HEART;

READ AT THE MEETING AT BRISTOL IN 1836.

[This report is rather lengthy, and there being little in it not previously ascertained in my experiments, or stated in the text, I only insert the *summary and conclusions.*]

1. The first sound of the heart, as heard in the chest, is generally complex in its nature, consisting of one constant or essential sound, and one perceptible only under certain

circumstances. This constant element of the first sound may be considered as intrinsic, appearing to depend on the sudden transition of the ventricles from a state of flaccidity in diastole to one of extreme tension in systole; while the extrinsic, or subsidiary sound, which in a variety of circumstances contributes largely to the first sound, arises from the impulse of the heart against the parietes chiefly of the thorax.

2. The collisions of the particles of the blood amongst each other, or against the interior parietes, valves, &c. of the heart, do not appear to have any share in the normal first sound of the heart; neither do the motions of the auriculo-ventricular valves;* and the attrition of the opposite interior surfaces of the heart's cavities seems purely hypothetical.

3. The principal, and apparently only cause of the second normal sound of the heart, is the sudden closure of the sigmoid valves, by the columns of blood that recoil back on them during the diastole, impelled by the elastic contraction of the arteries.

4. The columnæ carneæ appear to act simultaneously with the parietes of the ventricles, and in such a manner as to make it apparently impossible that the auriculo-ventricular valves should close with a flap, in the same manner as the sigmoid valves.—(Signed) C. J. B. WILLIAMS; R. B. TODD; J. CLENDINNING, *Reporter*.

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* [Although I signed the report, I considered this expression too strong, seeing that it was founded on negative results. Further experiments, performed the following year, convinced me that "the auricular valves, the cordæ tendineæ, &c. may have an equal share in the production of the first sound," as I had before expressed it, then doubtingly, because my experiments were negative on that point. Drs. Clendinning and Todd were both sceptical as to the sufficiency of muscular tightening as the cause of the first sound, until I showed them that similar, and even louder sounds, could be produced by sudden forcible contractions of the abdominal muscles. This experiment was repeated with the interposition of various solid bodies, and with the same result. This is a sufficient answer to Dr. Hope, who, having failed himself, in his last edition represents this experiment to be a mistake.]

SECOND REPORT
OF THE
LONDON COMMITTEE OF THE BRITISH ASSOCIATION FOR THE ADVANCEMENT OF SCIENCE,
ON THE
MOTIONS AND SOUNDS OF THE HEART;
COMMUNICATED AT THE MEETING AT LIVERPOOL,
SEPTEMBER, 1837.

The committee appointed in London to investigate the motions and sounds of the heart, have to present to the Association a short account of some investigations of the abnormal sounds of the heart and arteries, in which they have been recently occupied, and which were not comprehended in their former report.

Before describing these, the committee would remark, that although their inquiries have not been specially directed to that subject, yet they have had many opportunities of verifying the conclusions on the *natural* sounds, as presented in their report of last year; and these conclusions not having been since shaken by any experiment or rational objection, it may be considered as fairly established that the first, or systolic sound of the heart, is essentially caused by the sudden and forcible tightening of the muscular fibres of the ventricles when they contract; and that the second sound which accompanies the diastole of the ventricles, depends solely on the reaction of arterial columns of blood on the semilunar valves at the arterial orifices.

It further appears that the first sound may be increased by an additional sound of impulsion against the walls of the chest under certain circumstances of posture, of increased action of the heart, and of particular stages of the respiratory act. It is also obvious, that the character of the first sound may in some measure depend on the closure of the auriculo-ventricular valves, and on the quality of blood, inasmuch as these determine the nature and time of the resistance, against which the muscular fibres of the ventricles tighten. So likewise the vigour of the ventricular systole, the quantity of blood propelled by it, the sudden and complete character of the diastole, the fulness of the arterial tubes, as well as the perfect mobile and membranous condition of the semilunar valves, will determine the character and loudness of the second sound.

An experimental illustration of one of these conditions was observed by the committee during one of their experiments on the ass, in a great diminution of the second sound on the carotid artery being freely divided.

As additional illustrations of the production of a sound like that of the heart, by muscular contraction, the committee have noticed that accompanying the action of the panniculus carnosus of the ass during life, and the quivering contraction of muscles immediately after death. The sound produced in the latter case, in nature and frequency, closely resembled the first sound of the heart of the fœtus or of small animals. In investigating the morbid sounds of the heart, the attention of the committee has been chiefly directed to the causes of those remarkable and various accompaniments of the heart's action, called *murmurs*, which were happily compared by Laennec with the noises of blowing, filing, rasping, sawing, purring, cooing, &c. This inquiry consists of two parts: 1st, what is the essential physical cause of the phenomena in question? and 2nd, how does the apparatus of the circulation develope this cause in the various instances in which these phenomena are known to occur?

To the first of these inquiries the experiments of the committee supply what they trust will be deemed a satisfactory answer; the second is to be answered by extensive clinical and pathological observation, rather than by direct experiment; and although a few physiological experiments will be quoted to this point, yet the committee do not profess at present to do more than open this inquiry to all those who have the means of pursuing it.

Experiments on the production of Sound by the motion of Water through a Tube.

1. A caoutchouc tube, 18 inches long, and $\frac{3}{8}$ths of an inch in diameter, was attached to the stopcock of a reservoir in which there was water to the depth of from 8 to 10 inches.

When the water flowed perpendicularly through this tube, (the air being first expelled, and the lower end of the tube kept under water in the recipient below,) no sound was heard; but on pressing any part of the tube, so as to diminish its caliber, a blowing sound was heard at and below the point of pressure, and this sound became louder and more whizzing as the pressure was increased. The loudest sounds were obtained at the lowest end of the tube, where they were sometimes quite musical, and by increasing the pressure at regular intervals, a periodic increase and raising of the sound was produced, which closely resembled the sound heard in the neck, to which the French have given the name of "bruit de diable."

2. A pin being stuck transversely through the tube, a slight blowing was heard. A similar phenomenon more distinctly resulted from the use of a split goose-quill placed in the same way. A stronger blowing was produced by two threads across the diameter of the tube, especially when they were rather loose; and a still louder and shriller note ensued when a knot of string was fastened to the threads.

3. The same tube being adapted to the stopcock of a water supply-pipe, through which the current would be let to pass with great force, it was found possible to imitate every variety of blowing, whizzing, and musical murmurs, by varying the pressure on, or obstruction in, the tube, and by altering the force of the current. When the current was strong, the least obstruction caused a murmur, but, with weaker currents, greater obstructions became necessary for the same effect. An obstruction which, with a weak current, gave a blowing sound, produced with a stronger current a sound of a more whizzing character. Grating or rasping sounds were best obtained by the effect of a strong current on a knotted thread across the diameter of the tube. The musical or uniform sounds resulted from a moderately strong current through a considerable impediment; increasing the force of the current, or the degree of obstruction rendered them whizzing and imperfect; diminishing the current or the obstruction, converted them into a simple blowing.

When a sound was of an appreciable pitch, its note was higher in proportion to the force of the current and the amount of the obstruction—a fine forcible stream producing the highest note. Sometimes, however, with a strong current, a loud trumpet note would be set up, which was not altered in pitch, but only in force, by changing the strength of the current. This kind of note produced visible vibrations of the tube below the obstruction, and seemed to have relation to the length of the tube. In many instances these vibrations resembled closely the purring tremors and thrilling vibration sometimes felt in the region of the heart and large arteries. Musical sounds of a more variable character, like the cooing of a dove, the humming of an insect, or the whistling of wind, were produced with weak currents passing through a tube much obstructed. The pressure of a column of water, only two or three inches high, was sufficient to give acute whistling notes, which were sustained, although varying, even when the water that passed only fell in drops from the end of the tube.

4. Bending the tube to an angle produced a murmur; but no sound resulted from any curve that did not infringe on the caliber of the tube. A circular constriction, by a thread drawn round the tube, caused a murmur, which was blowing or whizzing according to the strength of the current.

5. The current issuing from the end of a tube, or from the mouth of an india-rubber bottle, produced a blowing sound when it impinged directly on an opposite surface, such as the side of the recipient or the end of the stethoscope; but unless the current were very strong, this sound was not produced when the current played on the surface very obliquely.

6. When the tube, with a weak current, was pressed on at two points, the murmur was heard at the point where the pressure was greatest; and by increasing the pressure at one point, the sound was stopped at the other. When the current was strong, it was easy, by a pretty equal pressure, to cause a murmur at both points.

7. With a strong caoutchouc tube, two feet long, and one inch in internal diameter, the same results were observed, but in a more remarkable degree, in consequence of the increased size of the tube. When the current was strong, and the pressure on the tube considerable, sounds were produced loud enough to be heard without applying the stethoscope to the ear, and the vibrations of the tube below the obstructions were so strong, that they threw the water in little jets from the outside of the tube.

8. In making the last experiment, the pressure of the water suddenly distended a portion of the tube into a globe about three inches in diameter, constituting a good representation of a circumscribed true aneurism. As long as the force of the current was sufficient to keep the walls of the dilated

portion distended, no murmur was produced; but when these walls became flaccid, the passing current caused a kind of dull fremitus in them. Slight pressure on the dilatation, or bending the tube to form an angle at the point, also sometimes occasioned a murmur.

9. A globular India-rubber bottle, three inches in diameter, being adapted to an aperture in the side of a tube, so as to form an elastic sac communicating with it; and the air being expelled, the current of water was directed through the tube. The same was done with a smaller tube, and a bottle one inch and a half in diameter. In some positions the current in passing the lateral sac occasioned a slight whizzing; but in others, as when the tube was straight, there was no sound. A sudden increase of current, or the removal of pressure from the sac, caused a whizzing by the entry of water into the sac. Independently of the current, sudden forcible pressure on the sac occasioned a whizzing with the expulsion of the fluid; and a similar whizzing attended its rapid reflux into the sac on the removal of the pressure.

10. Some of the preceding experiments being repeated with water made slightly glutinous with size, it was found that the various sounds were not quite so readily produced as with plain water, and required a greater force of current.

From all these results, (1, 2, 3, 4, 5, 6, &c.,) it is sufficiently plain that a certain resistance or impediment to a liquid current is the essential physical cause of all murmurs produced by the motion of fluids in elastic tubes. That any condition of the walls of the tube beyond the obstructing point is not, as it has been supposed, essential to the production of these sounds, is proved by the fact that they may be produced by an obstruction at the terminal orifice of the tube, or at the mouth of a gum elastic bottle, where there is no tube or wall beyond to cause them (1, 5); and usually this is the situation where they can be produced best, because here the current has acquired its greatest momentum, and finds a freer passage beyond the obstructed point. The more flaccid state of the portion of a tube beyond an obstruction is a necessary effect of the impediment caused by that obstruction to the passage of water. It is, therefore, a necessary concomitant of the obstruction, but it is not the cause of the sound. When, however, the sound occasioned by the obstruction is strong, its vibrations may be communicated to the whole contents and walls of the tube beyond, (3, 7,) which will then vibrate in system with it, and be capable of modifying its note; just as the tube of a reed instrument affects the note which is generated exclusively in the reed. On the other hand, when the sound is weak and varying, the condition of the tube or walls beyond it will not affect it.

In short, the laws of the production of sounds by liquids so closely resemble those which regulate the same phenomena in air, that it is unnecessary to enter into further detail respecting them.

It may be necessary to advert to an objection to this view, that a murmur is sometimes caused where there is no impediment to the course of a liquid, as when it passes suddenly from a small into a large tube, or into a sac. In the first place, it is not quite correct to assume that in this case there is no impediment, for the liquid in the large tube or sac, having less velocity than that in the small one, must itself be an impediment. But besides this, the course of the small swift current becomes changed by spreading into the larger channel, and instead of running smoothly parallel to the tube, now strikes its walls at an angle, causing a series of impulses and resistances, which, if forcible and rapid enough, constitute the vibration of sound. It is in the same way that a current produces a sound by impinging against an opposed surface. It may be observed, however, that these indirect kinds of impediment to a moving current are not so constantly attended with the production of sound as the direct obstacle presented by a narrowing of, or projection into, the caliber of the tube.

Experiments on the production of murmurs in the living body.

11. About two inches length of the common carotid artery of a young donkey was laid bare. Different degrees of pressure, either by the stethoscope, or by a probe pushed under the artery, occasioned a variety of murmurs, blowing, sawing, filing, and musical or cooing, at each pulse.

When the stethoscope was merely placed in contact without pressure, no murmur was heard, but only a simple impulse and sound, which was distinct only when the heart acted strongly.

12. The artery was scratched for a few seconds with the point of a scalpel; it gradually became sensibly smaller for the length of half an inch about that point. A strong solu-

tion of salt being applied, the contraction increased; but it was still of a gradual and tapering kind, and the stethoscope would not detect any murmur in it; very slight pressure was enough to cause a whizzing. The pulse at this contracted position was felt to be much harder and sharper than above or below it.

13. A small incision being made into the artery, a jet of blood issued, and a whizzing, sometimes continuous like the *bruit de diable*, sometimes only in pulses, was heard beyond the orifice, but no sound on that side of the orifice nearest the heart; the sound being, as usual, carried in the direction of the current. The incision being made larger, the blood spouted out, with whizzing, to the distance of more than six feet, and the animal died in the course of ten minutes.

After this last incision the beats of the heart were frequent, short, and pretty loud, but without a second sound, and without any murmur to the last. They continued for nearly two minutes after the respiration and consciousness had ceased, becoming gradually slower.

14. The committee repeated the observation that has often been made before, that a murmur can easily be produced by pressure on the subclavian, carotid, or femoral artery of the human subject. This murmur is generally of a grating or filing character, and is prolonged in proportion to the degree of pressure.

15. Whilst making the observations on the carotid, they found that a continuous murmur of very remarkable and variable characters could be produced by pressure on the jugular veins. The most common sound thus produced was like the humming of a gnat or fly, but occasionally it resembled the whistling of wind, the singing of a kettle, the cooing of a dove, and sometimes it was perfectly the remittent whizzing noise, which the French have called the *bruit de diable*. Dr. Ogier Ward, of Birmingham, had previously come to the conclusion that this sound is produced in the jugular veins, and the observations of the committee confirm this inference; but they do not agree with this physician in the opinion which he adopts from Messrs. Andral and Bouillaud, that the presence of these sounds *always* denotes a chlorotic state of the system, in which steel is indicated, or that they are essentially morbid symptoms at all. They may be produced in the healthiest subjects by moderate pressure applied to the lower part of the jugular veins, and are then found to

be modified by various circumstances, which can only affect the venous current. Thus they may be arrested or diminished by pressure on the vein above, by the horizontal posture, or hanging down the head, and by forced efforts to expire with the glottis closed. They may be restored in increased degree by suddenly desisting from any of these acts or circumstances.

The occasional pulsation or remittent character of these sounds seems to depend on the momentary increase of pressure caused by each pulse of the neighbouring artery; and when, as it sometimes happens, these pulses are attended with a whizzing, this is in a measure incorporated with the venous sound, and increases its periodic swell. The size and downward current of the jugular vein peculiarly adapt it for the production of sound, but it is probable that sounds may be produced by pressure on other veins, when circumstances accelerate the current within them. The committee have succeeded in detecting an obscure murmur in some of the large superficial veins in the arm and thigh. It is to be distinguished from muscular sound, which it resembles by its being heard only when the small end of the stethoscope is applied on a vein, and by its being stopped by pressure on that vein.

A louder continuous sound is sometimes heard at each side of the upper part of the sternum, which, from its resemblance to these venous sounds, and from its being stopped by forcible expiration, may be supposed to have its seat in the large venous trunks underneath.

Although it appears from these facts that the venous sounds are not necessarily signs of disease, yet the circumstance proved by the committee, (10,) that water is thrown into sonorous vibrations more readily than a liquid of a more glutinous character, renders it probable that these and other sounds depending on the motion of liquids in the apparatus of the circulation, may be more easily produced where the blood is thin and deficient in quantity; and under these circumstances they may occur in the neck, from the mere pressure of the muscles on the jugular veins.

The committee had planned several experiments for the further elucidation of the second part of the inquiry—namely, By what changes, functional and structural, does the apparatus of the circulation develope the physical causes of abnormal murmurs and sounds, in various instances in which they are known to occur?

Having failed in obtaining animals in time for this report, the committee propose to resume at a future time this part of the inquiry, so important for the elucidation of several obscure points in pathology, diagnosis, and practice, and to report the result of their labours at the next meeting, if the Association should think fit to re-appoint them for that purpose.

(Signed) C. J. B. Williams, M.D., F.R.S., &c. *Reporter.*
Robert B. Todd, M.D., *Professor of Physiology, &c. King's Coll. London.*

IV.—REPORT

OF

EXPERIMENTS ON THE CONTRACTILITY AND SENSIBILITY OF THE LUNGS AND AIR-TUBES,

BY CHAS. J. B. WILLIAMS, M.D. &c.

READ AT THE MEETING AT GLASGOW, SEPT. 1840.

When, at the request of the medical section of the British Association, I undertook, three years since, to investigate experimentally several doubtful points respecting the properties and offices of the apparatus of respiration in the higher classes of animals, I hoped to be able to include in the inquiry the chemical process of respiration and the vital effects of its interruption; but professional engagements have obliged me to relinquish these points. I have, consequently, restricted my attention to those more immediately bearing on practical medicine. The chief of these are the contractility and sensibility of the air-tubes and lungs.

It has long been a subject of controversy, whether the lungs and air-tubes are more than passive in the motions of respiration—if they possess any self-contracting or expanding power independent of the muscles which affect the capacity of the chest. Different writers, both ancient and modern, have maintained opposite opinions. Laennec, after Sennert, Bremond, and others, considered that the lungs have both a self-contracting and a self-expanding property: Haller, on the other hand, was led by experiment to deny that any independent rhythmical motions corresponding with the movements of respiration are exhibited by the lungs of animals, and Muller has confirmed this negative result. Nay, within the last few years, certain writers in this country and in France have denied altogether the muscular contractility of any part of the air-tubes. These negative observations are in opposition to the generally-received opinion, derived chiefly from the researches of Reisscissen, that the circular fibres of the air-tubes, from the trachea to their termination, are muscular. Very few attempts have been made to solve this problem by experiment. Wernier and Wedemeyer only succeeded in exciting partial contractions in the smaller bronchi; but, with all their results, Muller concludes that "it is remarkable that there exists at present no direct proof of the contractility of the muscular fibres of the trachea and its branches." (Elements of Phys. transl., by Baly.)

I need scarcely remark, that this is a subject of by no means merely speculative interest. Much of the pathology and treatment of various diseases of the respiratory organs, especially spasmodic asthma, is at present founded on the supposition of a muscular contractility of the air-tubes, the very existence of which is thus stated, by one of the most eminent of modern physiologists, to be little more than assumed.

The first objects of the following experiments were, by means of stimuli, electrical, chemical, and mechanical, to test the existence of muscular irritability in the air-tubes. In several of these experiments, which were performed at University College, I was kindly assisted by Professor Sharpey, Dr. Davison, Mr. James Blake, and several of my pupils, especially Messrs. Bloomfield, Carlile, Parkes, and Jos. King. In most of them a *hæmadynamometer*, or bent tube, like an inverted syphon, containing coloured liquid, was used: the short limb of this tube being adapted to the windpipe or one of its branches, the column of fluid within it would be readily moved by any contraction of the air-tubes or lungs causing pressure of the air on this column, and the rise or fall of the fluid was measured by a scale of inches and tenths.

Experiment 1. A dog was killed by pithing: the chest being immediately opened, the lungs became quite collapsed, and, with half the trachea, they were taken out, and the trachea tied to the brass tube of the hæmadynamometer. On completing the communication by turning the stopcock, the fluid in the tube oscillated several times to the amount of about $\frac{1}{4}$ inch. On passing a galvanic current (from a trough of 30 3-inch plates) from the margin of the lungs to the brass tube in the trachea, the fluid rose quickly but gradually nearly two inches; it sank speedily on breaking contact, again rose on completing it, but fell slowly when the contact was continued for some seconds. When the current was passed only through a single tube, the rise was still distinct, but much less in degree, not exceeding three or four tenths. The rise was repeatedly produced, but to a less extent, and in a little more than two minutes the galvanism seemed to have lost its effect.

2. The experiment was repeated with eight other dogs, of various sizes, with similar results. The column in one rose $2\frac{1}{2}$ inches. If the current was passed continuously for some length of time, the fluid in the tube fell, and could not be raised on the application of the galvanism for a minute or two after. It was found, also, that inflating the lungs impaired the effect of the current. On repeating the experiments further, it was found that the susceptibility was only exhausted for a time; for, on waiting two or three minutes between each application of the galvanism, the liquid was raised again and again for upwards of an hour—at first to the extent of one or two inches, but gradually to less, until it ceased to be affected. It was also found, that after inflating the lung, although no effect followed the application immediately after inflation, on waiting a minute or two, the usual result took place, but to a less extent, probably being diminished by the compressed state of the air, which therefore required more force to move it The fall on interrupting the current was pretty rapid, the fluid recovering its former level in from ten to twenty seconds.

3. Several trials were made to compare the contractility of the smaller and the larger bronchial tubes respectively. When the galvanic current was passed along the margins of the lobes, the liquid rose only two or three tenths, and soon subsided; but when the current was passed at right angles with larger tubes, there was more effect; but most when passed from the margin, or even the middle of the lobes, to the brass pipe of the hæmadynamometer, which was in the direction of the large air-tubes, the rise of the liquid was then from $\frac{1}{2}$ to $1\frac{1}{2}$ inches; and by repeating the contact after intervals of two or three minutes, these results continued

for more than an hour. On testing the contractility of small parts of the large tubes, it was found that the greatest extent of motion was produced on passing the current through the posterior part of the two great divisions of the trachea, where they subdivide into the large bronchi. In the trachea, and in the tubes below, the effect was to a smaller degree, but still well marked.

4. The preceding experiments were made on the lungs removed from the body immediately after death. I tried to obtain the same results with the lungs in the body, but it was not easy to galvanise the lungs without affecting the muscles of the chest, the contractions of which might interfere with the results. The expedient adopted was, immediately on the death of the animal, to adapt the hæmadynamometer to the trachea, then to open the chest and break back the ribs as far as possible, and, having separated the lungs from the walls of the chest by a piece of oil-cloth passed under them, then to apply galvanic wires to different parts of the lungs. This experiment gave less distinct results than when the lungs were removed from the body. It was repeated five times, but it will be sufficient to describe one in which the galvanism was tried on the par vagum, as well as on various parts of the lung. A large dog was pithed, and the hæmadynamometer tube adapted to the trachea. On opening the chest, the collapse of the lungs caused the fluid to rise three inches. The vagi in the neck were exposed. One was pinched—no effect on the fluid in the tube: the other was galvanised across a long portion of it—no effect on the bronchial tubes; but the œsophagus was strongly convulsed. On separating the nerve, and passing the current down it through the lungs, the fluid rose only about one-tenth, but on passing the current from the base of the lungs to the trachea, the fluid rose more than an inch, sinking again rapidly on withdrawing the galvanism.

In all the preceding experiments death was caused by pithing.

5. A rabbit was killed by a blow on the pole. The lungs being taken out and moderately inflated, the current was passed. Some doubtful rise at first, then, at three successive trials, a rapid fall of about one-tenth of an inch, followed by a rise. This was seen once when the galvanism was not applied, and seemed to depend on convulsive twitches of the irritable fibres, induced by the violent mode of death. About ten minutes after death the column had gradually sunk $\frac{1}{4}$ inch.

Galvanism then raised it slightly, and in a few minutes more the rise was repeatedly produced to the extent of two-tenths.

In the following the animal was killed by hæmorrhage.

6. A large dog was bled to death by dividing the jugular veins. Death preceded by very deep and rare breathing: the heart beat a few pulses after respiration and sensibility ceased. On adapting the hæmadynamometer to the trachea, the fluid remained stationary. On opening the abdomen, the column sank half an inch; on opening the chest, it rose five inches. After taking the lungs out of the body, galvanism failed to raise the column, but this failure probably arose from the tube becoming obstructed with viscid mucus; for, on applying the wires to the bifurcation of the trachea, the tubes were seen to contract most distinctly. This suggested another mode of observation, which I had mistrusted before, as liable to error. The lungs were cut by sharp scissors at right angles to the air-tubes, and the open sections of these being galvanised, contracted to half, and in some instances less than half, their former diameter. To ensure accuracy, the gaping ends, as soon as cut, were measured by compasses, or by tubes of similar size. The contraction was most distinct in the middle-sized tubes, about the size of a straw. These results were obtained for half an hour after death, and long after the heart had lost its irritability.

7. A large greyhound was killed by pithing. Lungs being taken out, one lobe was tried by galvanism and the hæmadynamometer, with the usual results. A large bronchus of another lobe was then cut longitudinally, laid flat, measured, and then galvanised: it became hollow, and contracted one-third, and at one part almost half. The divided ends of the other bronchi also contracted to less than half former diameter. In one instance, when the interior of a large bronchus was galvanised, bloody mucus was expelled from one of its branches, across which the current passed.

8. A bullock's lung was experimented on, about twenty minutes after death, which was caused, as usual, by a blow on the head and cutting the vessels of the throat. The bronchi were filled with bloody frothy mucus, and, on touching any of these with the galvanic wires, this froth exuded out. Their measured size also shrank under the same influence.

9. The same results were obtained with the lung of a calf, half an hour after death, caused, as usual, by bleeding and pithing. A small lobe of the lung, when galvanised,

also raised the fluid of the hæmadynamometer three-tenths.

10. Two years ago, I observed in the lungs of a horse, immediately after death by knacking, that the divided ends of the bronchi slowly contracted on being irritated with a scalpel; but as no measurement was used, and as those which were not so irritated also contracted, only more slowly, I did not then consider the result conclusive. It may, however, now be ranged with the other facts of the same kind.

11. In many instances, which I need not now detail, I tried to excite the contraction of the bronchi by chemical irritants, such as salt and diluted ammonia. I succeeded with the divided ends of the tubes; but it was more difficult to succeed with the dynamometer, it not being easy to introduce the irritant without mechanically affecting the dynamometer, and the irritant causing the formation of a mucous, viscid froth, which obstructed the tubes. The following was the most successful of these attempts.

12. A dog was pithed, and the lungs exposed by breaking back the ribs. To the trachea was tied a tube with two branches, furnished with stopcocks. One branch communicated with the hæmadynamometer; through the other the lungs were to be inflated, to give the tubes their full diameter, and make any contraction distinct. The irritating fluid was poured into the tube of the dynamometer in quantity sufficient that some might be driven into the trachea, by blowing into the upper end of the tube. The lungs being moderately inflated, the stopcock of the dynamometer was turned, when the column rose three inches; it was then depressed by blowing through the upper end, until a considerable portion of it dropped into the trachea. On withdrawing the mouth, the column was driven three-tenths above its former level, and slowly rose two-tenths more. This rise could only be ascribed to the contraction excited by the irritating liquid in the bronchi. By changing the position of the lung, several sudden starts of one or two tenths repeatedly occurred; probably dependent on the irritating fluid coming in contact with new portions of the tubes. But all these movements were much impeded by the viscid froth which collected in the bronchi. The lungs were now permitted to collapse, and the column subsided to a level. After a while it gradually sank two inches below the level, being probably thus drawn down by the re-expansion of the cartilaginous rings of the tubes, the irritability of the contracting fibres being ex-

hausted. On cutting into the lung, the trachea and bronchi were found unusually open, and filled with tenacious froth.

13. The experiment was repeated with saturated solution of common salt, with similar results.

14. The following experiment illustrates several of the same points, and further shows somewhat of the nature of the sensibility of the different parts of the air-passages.

Subject, a large Newfoundland dog. The trachea was exposed and divided below the cricoid cartilage. It was then held out by a hook. On being irritated with a scalpel, the posterior part gradually contracted until the rings met. The contraction was somewhat increased by galvanism; salt produced no further contraction. None of these irritations excited cough or other sign of uneasiness; but, on passing the finger upwards to the larynx, violent expiratory efforts were excited, and the glottis very forcibly closed upon the finger. These efforts forced a little bloody mucus out of the trachea. A scalpel was introduced into the trachea to its bifurcation, and made to scrape against the lining. No cough followed for some time; but about a minute after, on repeating the experiment, cough ensued, and increased on subsequent applications. In the upper part of the trachea, where the salt had been applied, there was a copious secretion of bloody mucus. The blood-vessels were now divided in the upper part of the thorax, and as the blood flowed freely, the inspirations became very deep and rare, and soon ceased, the heart's action continuing some seconds after.

The bronchi exhibited the full amount of contractility, one small lobe raising the dynamometer three-quarters of an inch. Sections also distinctly contracted on the application of salt, of ammonia, and of galvanism. One small bronchus, of the size of a crow-quill, became closed under the galvanic wire.

The upper part of the trachea, which had been irritated with salt, galvanism, and the scalpel, was found expanded to its greatest extent, while the part lower down was half contracted. Both contracted but slightly on being galvanised, but completely when the cartilaginous rings were divided.

Having satisfactorily proved the existence of irritable contractility in the air-tubes, I next proceeded to try how this was influenced by various medicinal agents which we are in the habit of using in the treatment of diseases of the respiratory apparatus.

15. A rabbit was poisoned by dropping some strong hy-

drocyanic acid into its mouth. It was seized in thirty seconds with convulsive and gasping breathing, with opisthotonic spasms, and died in ten seconds more. Galvanism produced no results with the dynamometer for the first five minutes; but after this it took effect, and raised the column first two-tenths, and in a quarter of an hour four-tenths. These effects gradually diminished, and ceased in half an hour.

16. About ten grains of extract of belladonna* were injected into jugular vein of a rabbit. In about fifteen seconds gaspings ensued, followed by slight convulsions, and death in less than a minute. On opening abdomen, heart seen through diaphragm acting quickly. The tube of the dynamometer being adapted to the trachea, the chest was opened, when the column rose immediately two inches, and slowly an inch more, when it remained stationary. Galvanism produced no effect at all for several minutes, and at last only a very feeble one, not one-tenth, with tube much inclined. Even this soon ceased, and no further effects could be obtained, although the experiment was continued for an hour.

17. A dog poisoned by mouth with hydrocyanic acid, fell down howling in half a minute, and ceased to breathe in about half a minute more. Heart continued to beat strongly after the chest was opened. Galvanism raised the column of dynamometer, adapted to trachea, two inches, and a diminishing effect continued half an hour. The same differences were observed in the contractility of different parts, as in the experiments after death by pithing; and the cut ends of the tubes contracted more than half an hour after death.

18. About twenty drops of strong hydrocyanic acid injected into carotid artery of an ass about six months old, immediately caused the breathing to become very deep and rare; but this effect passing off in about ten minutes, nearly a drachm more was injected. The deep convulsive breathing returned, accompanied by tetanic rigidity of the trunk and loud groaning. The breathing became very rare, whilst the heart beat violently and with a double second sound, and death ensued in about three minutes after second injection. A small lobe of one lung being galvanised raised the column of the dynamometer three-tenths repeatedly. Sections of tubes, in other parts, on being galvanised, contracted dis-

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* This and the other vegetable extracts were prepared by Mr. Squire, chemist to the Queen.

tinctly, but to a less extent than in the dog. Contractile motion distinctly seen on cutting a cartilaginous ring of the trachea, and then galvanising the musculo-membranous portion at the back.

19. About ten grains of extract of belladonna, mixed with water, were injected into the right jugular vein of a large dog. Pupils began to dilate in fifteen seconds, and breathing became more and more deep and laborious, and accompanied with howling and slight convulsive starts. The breathing ceased, and the eye became insensible, in two minutes after the injection. The integuments of the chest were then divided, which caused some flow of blood; the heart still beating, the animal, which had appeared to be quite dead, then made a gasp, and six or seven deep inspirations followed, during which the heart beat more strongly, and the eye again winked on being touched. This partial return of life soon ebbed again, and all motion ceased. On taking out the lungs, scarcely a trace of contractility could be detected in them by the dynamometer, and the cut ends of the tubes shrank but little on being galvanised. The trachea was quite relaxed, and very little approximation of the cartilages took place on the application of galvanism. The arteries also were less contracted than usual.

20. About thirty grains of extract of stramonium, mixed with water, were injected, at twice, into the cellular texture of the neck of a dog. Breathing soon became very quick, laborious, and occasionally stertorous. Lower extremities became very weak, and were dragged. Pupil extremely dilated, but sensibility of the eye and consciousness to sound remained till near death, which took place twenty-five minutes after second injection. The heart was found motionless and unirritable; right side and veins much engorged; arteries empty. On cutting and galvanising the bronchi, very little sign of contraction was perceived, and none on using the dynamometer. Œsophagus and intestines still irritable.

21. Two grains nitrate strychnia dissolved were injected into carotid of a large dog. In less than four seconds, tetanic convulsions came on, which continued one minute; the eye became insensible in half a minute. On opening body immediately, heart was motionless and unirritable; right side and veins enormously congested with black blood. The lungs being adapted to the dynamometer and galva-

nised, the column at first rose two-tenths, but not after. Voluntary muscles less irritable than usual.

22. Four grains nitrate strychnia dissolved were injected into jugular vein of a donkey two months old. In fifteen seconds spasms and tetanic rigidity. Eye soon lost its sensibility. Spasms of muscles of neck and back continued for more than a minute. On opening the chest the heart was still beating, the veins and right side much distended. The lungs being removed, and tried with dynamometer and galvanised, no sign of contraction was perceived, although the experiment was continued for some time.

23. Wishing to compare the contractility of the bronchi with that of the arteries, I made the following experiment. Immediately after the death of the subject of the preceding animal, ligatures were applied to the top of the descending aorta and to the left common iliac artery, and a dynamometer was adapted to the right iliac; some water, with a little soda dissolved, being first injected into the artery, to prevent the blood from coagulating. A galvanic current was then passed along the artery: the fluid of the dynamometer immediately began to rise, and rose steadily two inches. On stopping the current, the column continued to rise a few seconds, and then to sink progressively. After it had sunk about two inches, the galvanism again raised it two-tenths, but no more; and after this no further effect was produced. This was about twenty-five minutes after death. The auricles continued to be excited by galvanism more than half an hour after death, the contraction of one auricle being followed by that of the other. In this and another donkey, I had proved that the arteries contracted during life on the application of the galvanic stimulus, even more decidedly than with mechanical irritations.

24. Some tinct. lobeliæ, much concentrated by evaporation, was injected into the jugular vein of a dog. There were symptoms of uneasiness for a few minutes, but no further effect. A fluid extract obtained by evaporation ℥i.ss sol. bimeconate of morphia was then injected. In ten minutes peculiar convulsive starts, and some general tremor, with laborious breathing, followed by death in about one minute and a half. Heart continued to beat after chest was opened. Single lobes of the lungs being galvanised raised the dynamometer very slightly, not more than one-tenth. On cutting the lung, the sections contracted more distinctly,

but much less so than in other instances. The trachea was dilated.

25. About ʒss extr. conii, mixed with water, was injected into external jugular of a dog. In twelve seconds breathing became very quick and laborious, and in a minute the animal was dead. Bronchi not contractile as tested by the dynamometer, and less then usual tested by galvanising cut portions. The trachea was, however, so far contracted, that the end of the cartilaginous rings were in contact. The heart was still irritable.

I trust that many results of the preceding experiments are sufficiently evident without much further comment. Almost all of them prove that the air-tubes are possessed of irritable contractility, excitable by electric, chemical, and mechanical stimuli. The contractibility resembles that of the intestines, or of the arteries more than that of voluntary muscles or of the œsophagus, the contractions and relaxations being gradual, not sudden. They are, however, much less tardy than those of the arteries. The irritability of the contractile bronchial fibres is speedily exhausted by continued stimulation, and may be in some degree restored by rest, even in the lung removed from the body for an hour and more. The contractility of these fibres seems much influenced, in case of sudden death, by the mode of death, being for a short time suspended after death caused by a blow on the back of the neck, and sometimes by death caused by pithing, not by hæmorrhage. Several vegetable poisons destroy or impair this contractility. Extracts of stramonium and belladonna produced this effect most completely. Strychnia, extract of conium, and bimeconate morphia, also, to a great degree. Hydrocyanic acid, on the other hand, did not impair it at all. The action of these poisons on the bronchial fibres does not correspond with that on other contractile tissues, such as the heart and arteries, œsophagus and intestines, and the voluntary muscles; these in many cases having retained their irritability when that of the bronchi had been destroyed. It is possible that some medicinal agents (strychnia for example) may impair the contractility of the bronchial fibres by fixing them in a state of tonic spasm. The preceding experiments do not determine this point. The different state of the trachea, in some contracted, so that the ends of the cartilaginous rings overlap; in others expanded, so that they do not form three-fourths of the caliber of the tube, seems to countenance this supposition, and deserves further investigation. The bronchial fibres seem not to be excitable through the nerves of the lungs; for mechanical irritation of these nerves produced no effect on them, and passing the electric current through the nerves to the lungs was much less effectual in causing their contractions, than when the current was passed through the trachea.

THE END.

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